TASTE AND DEVELOPMENT

The Genesis of Sweet Preference

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
National Institutes of Health

Library
Forest Products Laboratory
Madison, Wisconsin

Acc, 1/12/78

TASTE AND DEVELOPMENT

The Genesis of Sweet Preference

Sponsored by

THE NATIONAL INSTITUTE OF DENTAL RESEARCH
and
THE JOHN E. FOGARTY INTERNATIONAL CENTER
FOR ADVANCED STUDY IN THE HEALTH SCIENCES

FOGARTY INTERNATIONAL CENTER PROCEEDINGS NO. 32

TASTE AND DEVELOPMENT

The Genesis of Sweet Preference

Edited by

JAMES M. WEIFFENBACH, Ph.D.

National Institute of Dental Research

DHEW Publication No. (NIH) 77-1068

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service National Institutes of Health
Bethesda, Maryland

1977

TASTE AND DEVELOPMENT

The Genesis of Sweet Preference

Edited by

JAMES M. WEIFFENBACH, Ph.D.

National Institute of Dental Research

Sponsored by

THE NATIONAL INSTITUTE OF DENTAL RESEARCH
and
THE JOHN E. FOGARTY INTERNATIONAL CENTER
FOR ADVANCED STUDY IN THE HEALTH SCIENCES

FOGARTY INTERNATIONAL CENTER PROCEEDINGS NO. 32

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, D.C. 20402
Stock Number 017–047–00009–4

CONTRIBUTORS

Robert M. Bradley, B.D.S., M.S.D., Ph.D.

Department of Oral Biology
School of Dentistry
University of Michigan
Ann Arbor, Michigan

George Collier, Ph.D.

Department of Psychology
Rutgers University
New Brunswich, New Jersey

Charles Crook, Ph.D.

Walter S. Hunter Laboratory of Psychology
Brown University
Providence, Rhode Island

Jeannette Desor, Ph.D.

Monell Chemical Senses Center
University of Pennsylvania
Philadelphia, Pennsylvania

Cynthia DuBose, B.A.

U.S. Army Natick Development Center
Natick, Massachusetts

Trygg Engen, Ph.D.

Walter S. Hunter Laboratory of Psychology
Brown University
Providence, Rhode Island

John L. Falk, Ph.D.

Department of Psychology
Rutgers University
New Brunswick, New Jersey

Marion Frank, Ph.D.

Rockefeller University
New York, New York

Bennett G. Galef, Jr., Ph.D.

Department of Psychology
McMaster University
Hamilton, Ontario
Canada

Lawrence S. Greene, B.A., D.M.D., Ph.D.

Monell Chemical Senses Center
University of Pennsylvania
Philadelphia, Pennsylvania

Joel Grinker, Ph.D.

Rockefeller University
New York, New York

Patricia Henderson, M.A.

Department of Psychology
McMaster University
Hamilton, Ontario
Canada

Edward Hirsch, Ph.D.

Department of Psychology
Mount Holyoke College
South Hadley, Massachusetts

Katherine A. Houpt, V.M.D., Ph.D.

New York State College of Veterinary Medicine
Cornell University
Ithaca, New York

T. Richard Houpt, V.M.D., Ph.D.

New York State College of Veterinary Medicine
Cornell University
Ithaca, New York

Harry L. Jacobs, Ph.D.

U.S. Army Natick Development Center
Natick, Massachusetts

Norge W. Jerome, Ph.D.

Department of Human Ecology and Community Health
University of Kansas Medical Center
Kansas City, Kansas

Allan Jessup, B.A.

Department of Psychology
McMaster University
Hamilton, Ontario,
Canada

Paul Johnson, M.D.

Nuffield Institute for Medical Research
University of Oxford
Headington, Oxford
England

Seymour Kreshover, D.D.S., M.D., Ph.D.

National Institute of Dental Research
National Institutes of Health
Bethesda, Maryland

Rachel H. Larson, Ph.D.

Preventive Methods Development Section
National Caries Program
National Institute of Dental Research
National Institutes of Health
Bethesda, Maryland

Milo D. Leavitt, Jr., M.D.

Fogarty International Center,
National Institutes of Health
Bethesda, Maryland

Jacques Le Magnen, Sc.D.

Laboratoire de Neurophysiologie Sensorielle et Comportementale
Collège de France
Paris, France

Lewis P. Lipsitt, Ph.D.

Walter S. Hunter Laboratory of Psychology
Brown University
Providence, Rhode Island

Owen Maller, Ph.D.

Monell Chemical Senses Center
University of Pennsylvania
Philadelphia, Pennsylvania

Herbert Meiselman, Ph.D.

U.S. Army Natick Development Center
Natick, Massachusetts

Charlotte Mistretta, Ph.D.

Department of Oral Biology
School of Dentistry
and
Center for Human Growth and Development
University of Michigan
Ann Arbor, Michigan

G. Rolfe Morrison, Ph.D.

Department of Psychology
McMaster University
Hamilton, Ontario
Canada

Howard Moskowitz, Ph.D.

U.S. Army Natick Development Center
Natick, Massachusetts

Hans R. Mühlemann, M.D., D.D.S.

Department of Cariology and Periodontology
University of Zurich
Dental Institute
Zurich, Switzerland

Shizuko Muto, Ph.D.

Graduate School of Home Economics
Japan Women's University
Takata-Toyokawa-cho, Bunkyo-ku
Tokyo, Japan

Geoffrey Nowlis, Ph.D.

Rockefeller University
New York, New York

Carl Pfaffmann, Ph.D.

Rockefeller University
New York, New York

Curt P. Richter, Ph.D.

The Henry Phipps Psychiatric Clinic
Johns Hopkins University
Baltimore, Maryland

Judith Rodin, Ph.D.

Department of Psychology
Yale University
New Haven, Connecticut

D. M. Salisbury, M.B., B.S.

Department of Pediatrics
University of Oxford
Oxford, England

Grant K. Smith, Ph.D.

Department of Psychology
McMaster University
Hamilton, Ontario
Canada

Edwin Smutz, Ph.D.

Army Research Institute
Fort Hood, Texas

Walter C. Stanley, Ph.D.

Section on Comparative Biopsychology
Laboratory of Brain Evolution and Behavior
National Institute of Mental Health
National Institutes of Health
Bethesda, Maryland

Eliot Stellar, Ph.D.

Institute of Neurological Sciences
University of Pennsylvania
Philadelphia, Pennsylvania

Jacob Steiner, Ph.D.

Hebrew University
Hadassah School of Dentistry
Jerusalem, Israel
and
University of Pennsylvania
Philadelphia, Pennsylvania

Dean Alan Thompson, M.S., M.D.

Monroe Community Hospital
Endocrine and Metabolism Unit
Rochester, New York

Richard L. Webber, D.D.S., Ph.D.

Clinical Investigations Branch
National Institute of Dental Research
National Institutes of Health
Bethesda, Maryland

James M. Weiffenbach, Ph.D.

Oral and Pharyngeal Development Section
National Institute of Dental Research
National Institutes of Health
Bethesda, Maryland

Paul Thomas Young, Ph.D.

University of Illinois
Champaign, Illinois
Retirement address
573 South Boyle Avenue
Los Angeles, California

PREFACE

Sweet is a modality of oral chemosensitivity and also the example *par excellance* of hedonic experience. The human preference for sweetness plays a critical role in the regulation of dietary intake and thus in the maintenance of health. Modern food processing and marketing methods increasingly substitute sugar for other less sweet dietary carbohydrates and result in increased per capita consumption of sugar. Correlational studies implicate these dietary changes in the rising incidence of heart disease, diabetes and obesity. Animal experimentation and well controlled clinical observations of humans show a clear casual relation of sugar ingestion to dental caries.

The sensory, perceptual and motivational phenomena of taste are under extensive and productive investigation in mature animals and man. Much less is known about the taste sensitivities and preferences of immature organisms. Developmental studies of other modalities (e.g. vision, audition and pain) have contributed to the understanding of adult perception and specifically demonstrated the critical importance of early sensory experience for adult perception. Developmental studies of taste should likewise contribute to the understanding of taste in the adult and might well provide insights into the role of early taste experience in the generation of the stable but destructive eating patterns of adults in our culture. This conference and the book derived from it offer such a developmental perspective to the study of preference for sweet in man and, potentially, to the devising of methods or routines for the prophylaxis of disease through the modification of dietary patterns.

The Conference on the Development of Sweet Preference was held June 19–21, 1974 under sponsorship of the John E. Fogarty International Center and the National Institute of Dental Research. The chapters of this volume were prepared in retrospection of the conference presentations. The discussion contributions were edited by the participants.

INTRODUCTION

Milo D. Leavitt, Jr.

It is a great pleasure to welcome you to the National Institutes of Health and to the Fogarty International Center.

In the course of planning for today's conference, it was suggested that I take a few moments this morning to tell you about the Fogarty International Center and its origins. The Center was established on July 1, 1968 as a memorial to the late Congressman John Fogarty who for a period of twenty years championed the expansion of this nation's health research programs. It had been Mr. Fogarty's hope for a number of years that an international center for advanced study might be created on the NIH reservation with the general objective of relating the development of new knowledge through medical research to the larger questions of science and society. Shortly after Mr. Fogarty's death in 1967, Mr. Melvin Laird, who had served with John Fogarty on the House Appropriations Subcommittee, introduced a proposal in Congress which established the Center as a new program activity at the National Institutes of Health.

I would like now to describe very briefly our advanced study program at the Center. It consists of two essential elements. The first of these is the Scholars-in-Residence Program which permits us to bring authorities from the United States and abroad to the National Institutes of Health for varying periods of time, several months to a full academic year. While the Scholars are at NIH they may engage in a variety of activities which range from the conduct of seminars involving our intramural scientists to the preparation of scientific monographs and textbooks. The other very important activity within the advanced study program is the Conference and Seminar Program. Through this program we are able to mount international conferences, workshops, and small seminars devoted to major problems in science and medicine. At this point, I would like to emphasize that the Fogarty International Center provides a forum which attracts both American and foreign scientists. We make a conscious effort to assure that there is a proper mixture of U.S. and foreign scientists who participate in our advanced study program.

Next, I should like to say a few words about our program interests in the Center. We are giving major emphasis to problems of disease prevention, to the problems of environment, and to recent advances which have been made in human genetics. We are also very much concerned with the problems which now confront medical education in the United States and in the advanced countries of the world. We shall pursue these topics for a number of years since we are well aware that issues of such importance to modern society will require long-term study.

In addition to these independent program interests, we are eager to collaborate with the other Institutes here at the National Institutes of Health in the support of conferences devoted to biomedical topics which are of high program relevance to the NIH and the Department of Health, Education, and Welfare. Today's conference on the development of sweet preference is an excellent example of the joint efforts of the National Institute of Dental Research and the Fogarty International Center to foster interest in new and developing areas of biomedical research. May I wish you a most successful meeting.

OPENING REMARKS

Seymour J. Kreshover

Whenever, from around the world, a small group of investigators can be brought together to describe their individual journeys into unknown regions of a common interest and to speculate on the significance of their findings, they invariably encourage and inform each other. Moreover they re-examine the significance of findings ignored or misunderstood, and inspire further journeys along different paths.

Of the many interesting aspects of dental research, this conference on the Development of Sweet Preference exemplifies perfectly the cooperative, mutually stimulating, generative nature of science.

The mouth is a complex organ system essential for life, and, upon it, physical and mental well being depend to a far greater extent than is generally recognized. For many practical aspects of oral health and disease, taste preference, whether congenital or acquired, could be pivotal. Accordingly, the subject deserves, and eventually will repay, this attention from dentists, physicians, and other professionals of diverse scientific backgrounds.

One of the special privileges of serving at the National Institutes of Health is that the Fogarty International Center is invaluable in affording a mechanism for mutually advantageous cooperation between investigators in the United States and abroad for the purpose of lighting the path of biomedical research. For its role in the planning and conduct of this conference, we express our sincerest appreciation.

CONTENTS

Page

Contributors vii
Preface xi
Introduction—Milo D. Leavitt, Jr. xiii
Opening Remarks—Seymour J. Kreshover xv

PART I
CONTRIBUTIONS FROM THE ELECTROPHYSIOLOGY AND PSYCHOPHYSICS OF TASTE

Chapter
1. Biological and Behavioral Substrates of the Sweet Tooth
Carl Pfaffmann 3
Discussion: *Le Magnen, Jacobs, Steiner, Rodin, Collier* and *Richter* 21
2. The Distinctiveness of Responses to Sweet in the Chorda Tympani Nerve
Marion Frank 25
Discussion: *Mistretta, Bradley, Le Magnen, Grinker, Pfaffmann* and *Weiffenbach* 39
3. Does Saccharin Have a Dual Taste for the Rat?
G. Rolfe Morrison and *Alan Jessup* 42
Discussion: *Collier, Jacobs, Mistretta* and *Nowlis* 47

PART II
RESPONSES TO SWEET IN THE FETUS AND SUCKLING YOUNG OF MAMMALS OTHER THAN MAN

4. Taste in Utero: Theoretical Considerations
Charlotte M. Mistretta and *Robert M. Bradley* 51
Discussion: *Stanley, Johnson, Collier, Lipsitt, Weiffenbach, Pfaffmann, Nowlis, Desor, Webber, Grinker* and *Galef* 64
5. Developmental Changes in Rat Fungiform Papilla Populations
P.W. Henderson and *G.K. Smith* 70
Discussion: *Mistretta* 85

6. The Neonatal Pig: A Biological Model for the Development of Taste Preferences and Controls of Ingestive Behavior
Katherine A. Houpt and *T.R. Houpt* 86
7. Comparative Observations on the Ontogeny of Taste Preference
Harry L. Jacobs, Edwin R. Smutz and *Cynthia N. DuBose* 99

PART III
SWEET SENSITIVITY AND PREFERENCE IN HUMAN NEWBORNS

8. Preliminary Studies on Feeding and Breathing in the Newborn
Paul Johnson and *D.M. Salisbury* 111
Discussion: *Mistretta, Pfaffmann* and *Nowlis* 123
9. Taste in Human Neonates: Its Effect on Sucking and Heart Rate
Lewis P. Lipsitt 125
Discussion: *Collier, Maller, Steiner, Johnson* and *Le Magnen* . 141
10. The Discrimination of Glucose and Water by Human Infants
Trygg Engen 143
11. Taste and the Temporal Organization of Neonatal Sucking
C.K. Crook 146
Discussion: *Steiner, Collier* and *Nowlis* 158
12. Preference for Sweet in Humans: Infants, Children and Adults
J.A. Desor, Owen Maller and *Lawrence S. Greene* 161
13. Facial Expressions of the Neonate Infant Indicating the Hedonics of Food-Related Chemical Stimuli
Jacob E. Steiner 173
Discussion: *Moskowitz* and *Pfaffmann* 188
14. From Reflex to Representation: Taste-elicited Tongue Movements in the Human Newborn
Geoffrey H. Nowlis 190
Discussion: *Le Magnen* and *Steiner* 203
15. Sensory Mechanisms of the Newborn's Tongue
James M. Weiffenbach 205
Discussion: *Pfaffmann* 212

PART IV
TASTE EXPERIENCE AND THE DEVELOPMENT OF A DIETARY PREFERENCE FOR SWEET

16. Mechanisms for the Transmission of Acquired Patterns of Feeding From Adult to Weanling Rats
Bennett G. Galef, Jr. 217
Discussion: *Nowlis, Stanley, Le Magnen* and *Jacobs* 232
17. Taste Experience and the Development of a Dietary Preference for Sweet in Humans: Ethnic and Cultural Variations in Early Taste Experience
Norge W. Jerome 235
Discussion: *Nowlis, Moskowitz, Collier, Jacobs, Grinker, Rodin, Mistretta* and *Pfaffmann* 246
18. Dietary Sweet: Exposure and Preference Among Japanese Children and in Laboratory Rats
Shisuko Muto 249

PART V
RELATING TASTE TO INGESTION

19. The Role of Sweetness in the Food Preference of Young Adults
Herbert L. Meiselman 269
Discussion: *Morrison, Engen, Rodin, Galef, Grinker* and *Collier* 279
20. Sensations, Measurement and Pleasantness: Confessions of a Latent Introspectionist
Howard R. Moskowitz 282
21. Implications of Responsiveness to Sweet Taste for Obesity
Judith Rodin 295
Discussion: *Pfaffmann* and *Le Magnen* 308
22. Effects of Metabolic State on Taste Parameters and Intake: Comparisons of Human and Animal Obesity
Joel A. Grinker 309
Discussion: *Jerome, Moskowitz, Stellar, Engen, Thompson* and *Le Magnen* 327
23. Nutrient Factors as Determinants of Sucrose Ingestion
George Collier and *Edward Hirsch* 330
24. Sugar Ingestion and Caries
Rachel H. Larson 345
Discussion: *Stellar, Pfaffmann, Mühlemann* and *Jacobs* 350

PART VI
COMMENTS AND PERSPECTIVES

25. Sweet Preference and the Sensory Control of Caloric Intake
Jacques Le Magnen 355
26. Sweet Preference and Hedonic Experience
Eliot Stellar 363
27. Sweet Abuse and the Addiction Model
John L. Falk 374
28. Six Common Sugars as Tools for the Study of Appetite
Curt P. Richter 387
29. The Role of Hedonic Processes in the Development of Sweet Taste Preferences
Paul Thomas Young 399

Author Index 419
Subject Index 427

PART I
CONTRIBUTIONS FROM THE ELECTROPHYSIOLOGY AND PSYCHOPHYSICS OF TASTE

Chapter 1

BIOLOGICAL AND BEHAVIORAL SUBSTRATES OF THE SWEET TOOTH

Carl Pfaffmann

I would like to begin with a quote from Hubert Frings [13] written in 1946 when he was in the Department of Biology at Gustavus Adolphus College:

> The production of pure sucrose, which modern man uses to satisfy his "sweet tooth" is a scientific achievement. This does not mean, though, that a predilection for sweet foods is limited to civilized man. In fact, the "sweet tooth" is not even limited to man. So far as I can discover there is no animal which will not accept sugar, and many which actually take sweet things by preference.
>
> The acceptance of—or even preference for—sweets by horses, dogs, mice, rats, and other domesticated and domestic mammals is very well known. That this is not due entirely to their close association with man is suggested by the fact that many wild mammals are also known to be fond of sweets: bears, raccoons, skunks, and others.
>
> Another group of animals which has many members commonly known to take sweets is the insect. What camper has not wished that flies, ants, wasps, or bees were not so readily attracted by sugar? Besides these picnic pests, crickets and butterflies are commonly known to seek sweets. An uncapped jar of syrup will trap thousands of ants which drown in their efforts to get the sticky prize. . . .
>
> To start with the Vertebrates, rabbits, chickens, turtles and frogs may seem fairly unlikely as possessors of a "sweet tooth," and yet they do accept sucrose as food. With rabbits and chickens, for instance, sugar-water, in all concentrations above the lowest they accept, is taken when the animals have all the water they want. This shows that these animals can distinguish sugar-water from pure water, and that they accept it as food.

The question of whether chickens or other birds have a sweet tooth has been a debatable point. Morley Kare [21] has emphasized that although many birds give evidence of a taste sensitivity only a few including parrots and hummingbirds show a sweet preference. However, Jacobs [19] cites Engelmann and his own work giving evidence for sweet preference in chicks. Frings [13] notes that when birds have all the water they want, they may show a preference for sugar-water in concentrations above threshold.

Acknowledgment: This research was supported by Grant No. BMS 75–18067 from the National Science Foundation.

Anatomically many birds have only perhaps forty or thirty taste buds, very few compared with other vertebrates particularly mammals.

I accept Kare's objections to a blanket assumption of a "sweet tooth" in all living organisms, but to me it is much more impressive that there are so many creatures who prefer and accept sugars. When in doubt, one is more likely to be right than wrong in expecting an organism to show a preference for sugar and related substances.

Man's nearest evolutionary relatives, present-day primates, also display the preference for sweet. In *The Evolution of Primate Behavior* Jolly [20] notes that most primates are omnivorous eating fruit, leaves, insects, and even meat when they can get it. Vegetable food, however, is the staple for most species and meat eating, generally, is rare except by baboons and chimpanzees. The bulk in the primate diet comes from plants. Some species eat chiefly fruit, others largely leaves, and some can subsist, if necessary, on grass. Spider monkeys not only eat, but prepare the fruits of Tocoyena pittieri which hang from stout stems and are protected by a thick green skin. The monkey bites through the skin of a fruit, but, finding the pulp green and bitter, leaves it alone. When the troop returns a few days later it is full of a sort of sweet jam; opening the fruit has accelerated its ripening, and the monkeys appreciatively scrape it out to the last drop.

Bicchieri [4] in *Hunters and Gatherers Today*, notes: "For more than 99 percent of the approximately two million years since the emergence of a recognizable human animal, man has been a hunter and gatherer." One form of gathering in neolithic times is shown in the cave painting of Figure 1–1. "Living in the sedentary, densely populated communities made possible by the adoption of agriculture as a life base is a new condition to which man has only begun to make physiological and psychological adjustments. The shift from hunting and gathering to agriculture, and finally to an industrial base, is so recent that today there are still human communities living within the framework of each adaptation. The number of hunting and gathering communities is dwindling rapidly. This life form will be extinct within a few more years." They are now being actively investi-

Figure 1–1. Cave painting from Arana cave at Bicorp near Valencia in Spain of a neolithic man robbing a store of wild honey. (Reproduced from Deerr [8].)

gated before they die off or become integrated into a new life style.[22]

The Pitjandjara [35] are one such hunting and food-gathering aboriginal people of the Great Western Desert of Australia. The climate is arid, but summer rains replenish the water storage. There is a mixture of vegetation and grasses on the rocky slopes and low hills, sandy banks, and dunes. The outcroppings may have a few native fig trees. One important food source for these people—the women do most of the gathering and the men do the hunting—are lerp scales, a sweet waxy secretion produced by the young plant as a protection against the jumping lice and related genera. The scale occurs on several species of Acacia and Eucalyptus and provide a source of sugar upon which they may need to feed. The Wama Wanari, another lac scale, also infests large areas of mulga, another Acacia species. The Pitjandjara may live for days on these substances, breaking off the twigs covered with scale and drawing them through their lips, often made sore and bleeding by doing so. They will tolerate pain and discomfort for sugar.

Honey ants occur in the mulga scrub. After heavy rains, the ants harvest the flow of honey from flowers and the sweet sap from glands on the growing tips of mulga twigs. This the ants store well below ground level in the inflated abdomens of certain worker ants, each the size of a pea. These living stores of honey tide the ants over the periods when lack of rain prevents the flowering of the mulga. But these honey stores are also one of the buffer food supplies for natives in dry years. Usually women dig for honey ants using a digging stick and wooden scoop. Sometimes, when other food is in short supply, men join in the search. Holes as deep as three and four feet and several yards in diameter may develop as they search for storage holes with their living sacs of sweetness. There is a totem and mythology of an ancestral being, the original honey ant, the Purara, that came into existence at what is now a ceremonial place. These evolutionary and anthropological examples, I trust, will give us a broader view of the processes under consideration in this symposium.

Now to the mechanism of the sensory apparatus that guides the search of animals and man for sweetness. Figure 1–2 displays scanning electron micrographs of the tongue made at Tallahassee by Graziadei and Beidler showing vallate, foliate and fungiform papillae surrounded by nongustatory papillae. Gustatory stimulation takes place through the pore seen in the top figures. Figure 1–3 is a more traditional photomicrograph showing single taste buds. These happen to be located, as a matter of fact, not in the fungiform papillae of the tongue but on the roof of the rat's mouth in the vicinity of the nasoincisor duct connecting the oral and nasal cavities. Dr. Curt Richter, a number of years ago, called my attention to the fact that taste buds existed on non-lingual regions of the mouth. The taste buds in the figure are located on the oral aspect of the hard palate in the forward portion of the mouth. Lingual taste buds are distributed on the front of the tongue in fungiform papillae and on its back in slit-like foliate papillae at the sides and the single vallate papilla on its dorsal surface (See Figure 1–4).

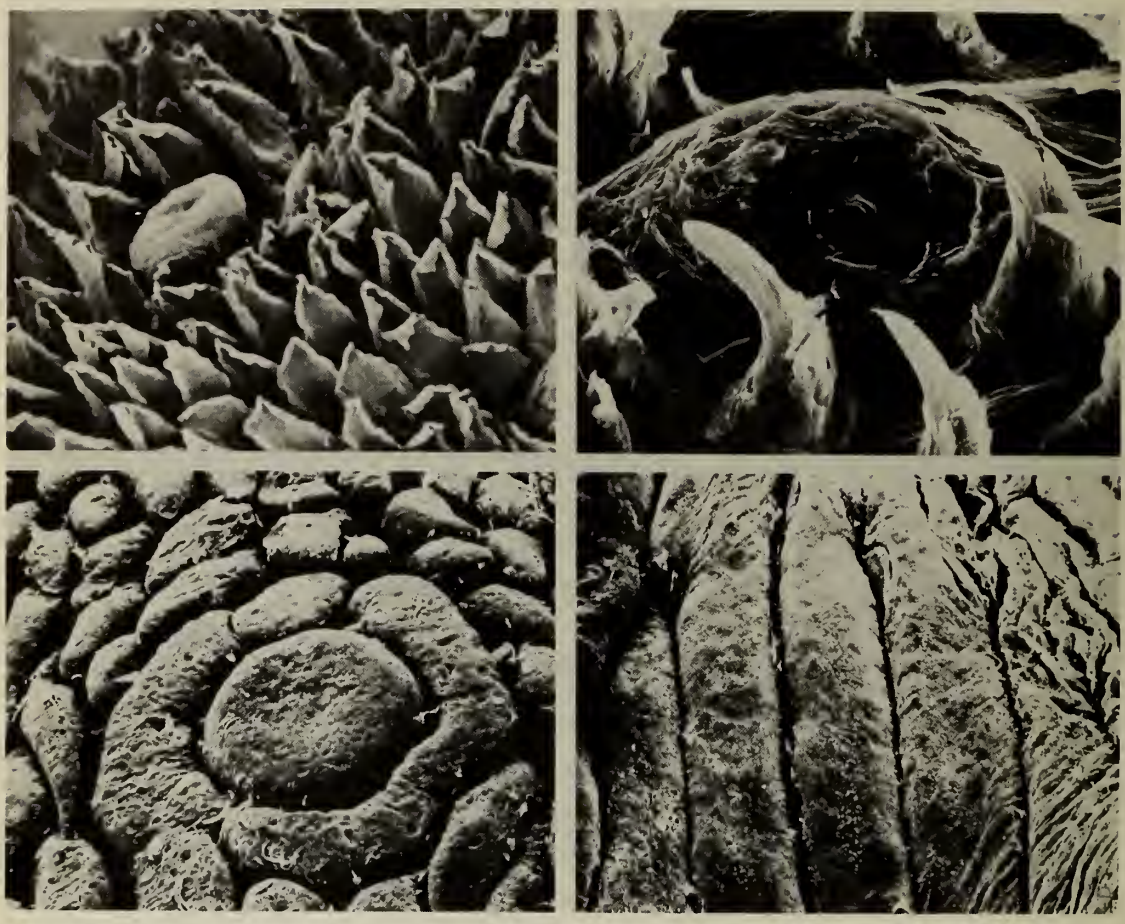


Figure 1–2. Scanning electron micrographs of a single fungiform on rabbit tongue (top left) and rat tongue (top right); circumvallate papilla of 3-week puppy (lower left) and foliate papillae of rabbit (lower right). (Reproduced with permission; Beidler [A].)

Figure 1–5 illustrates the electrophysiological method applied to the chorda tympani nerve showing the multi-fiber discharge as an asynchronous display. To make this recording somewhat more meaningful an index of sensory stimulation is provided by a quantitative write-out of the amount of the activity of the total nerve. Such recordings reflect events at the receptor cells with which chorda tympani nerve terminates.

The chorda tympani supplying the front of the tongue is the most convenient and the most widely used nerve for taste studies because it is readily accessible and contains very few sensory fibers other than taste. The ninth nerve innervating the back of the tongue is only recently getting attention. There is much to be done with it because of its very important location close to the entrance of the digestive tract. There is the possibility

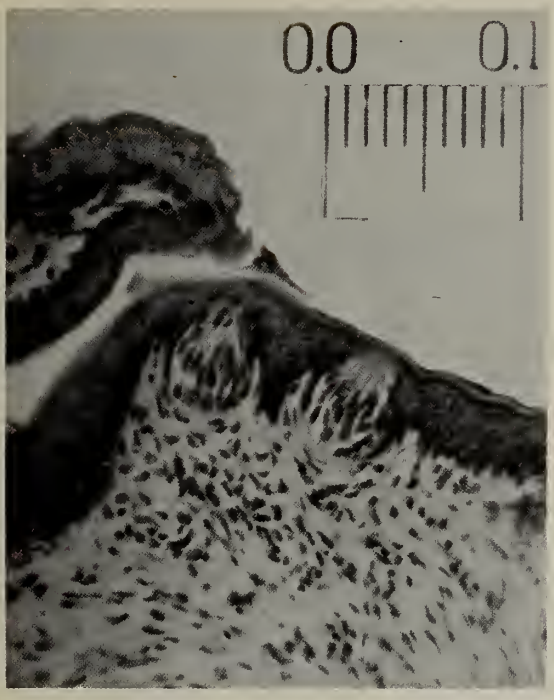


Figure 1–3. Photomicrograph of two taste buds near naso-incisor duct on roof of rat's mouth. Ten divisions equal 0.1 mm. (Reproduced with permission; Pfaffmann [D].)

that these two receptive fields actually tap separate reflexive areas—that the modalities contained therein exert specific stimulus control—in consummatory swallowing reflexes which

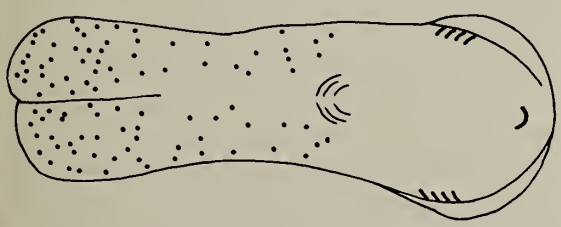


Figure 1–4. Tracing of rat's tongue showing location of fungiform, foliate, and the single vallate papillae. (Courtesy of Dr. Curt Richter; Based on Fish, Malone & Richter [B].)

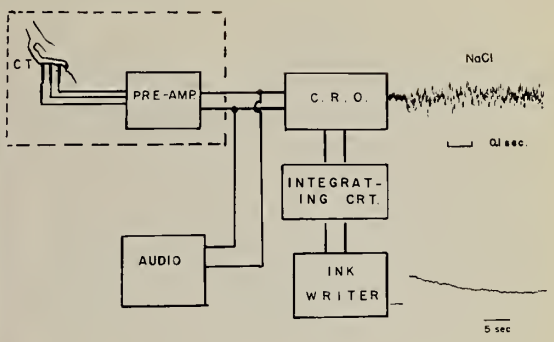


Figure 1–5. Schematic of electrophysiological recording of taste activity in chorda tympani nerve. (Reproduced with permission; Pfaffmann [E].)

Dr. Nowlis will discuss in his presentation. This recording method has been applied to man by Diamant, et al.[11] in the course of surgery on the middle ear making possible direct correlations with human sensations. After applying gymnemic acid, a specific blocking agent of the sweet taste (Warren & Pfaffmann [36]) the Swedish investigators have shown that the responses to sugar and saccharin in the human chorda reduce to zero but the responses for salt, acid and bitter remain intact. Gymnemic acid will have a similar effect on certain animal species: dog, hamster, and macaque monkey, but not in many others. The reasons for these species-specific exceptions are not yet clear. The combined recording and psychophysical observations in man gives additional assurance that the chorda tympani response does indeed provide a measure of taste sensation and indeed makes possible the logical jump to inferring a "sweet taste" in animals.

Figure 1–6 is a composite graph based on Dr. Frank's work.[29] Comparing the glossopharyngeal with the

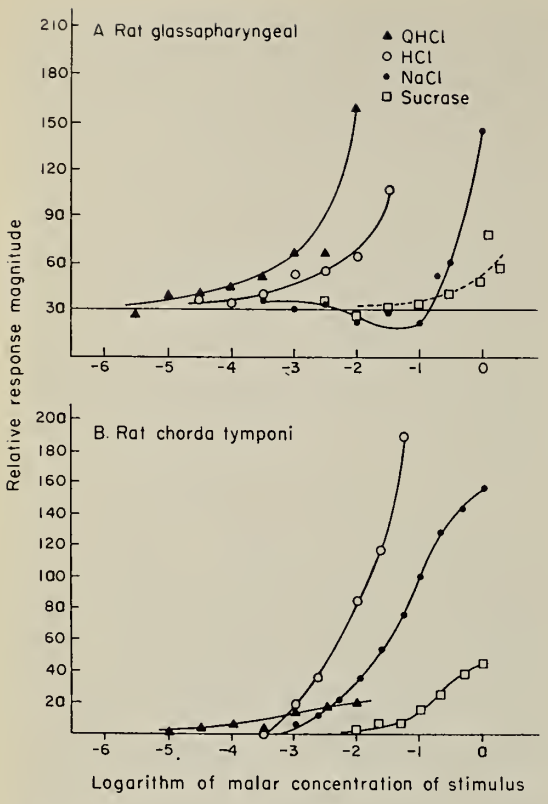


Figure 1–6. Relative magnitude of neural response to infusion into the circumvallate papilla (A) and to stimulation of the anterior portion of the tongue (B) in rat. (Reproduced with permission; Pfaffmann, Fisher, & Frank [29].)

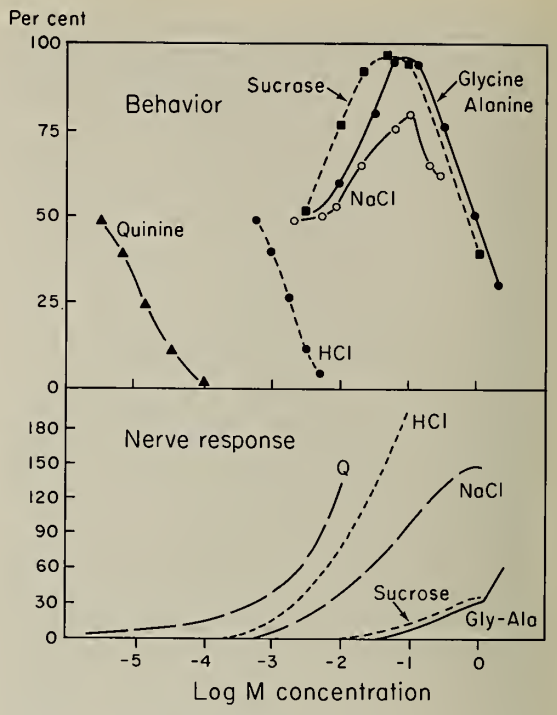


Figure 1–7. Behavioral preferences and aversions to taste stimuli (upper panel) and composite VII–IX nerve taste discharges (lower panel). Glycine and alanine derived from Halpern, Bernard, & Kare [C].

chorda tympani shows a remarkable quinine sensitivity in the vallate as contrasted with either the foliate or fungiform papillae. Quinine on the front is not much above noise level. Another interesting point is the relatively small sodium chloride response at the back except for the higher concentrations which behaviorally are avoided. In the front, of course, NaCl is a very good stimulus; HCl is good but not so different in the three receptor fields. Sugar is not remarkable either on the front or on the back of the rat tongue. This is surprising in view of the rat's good behavioral preference for sugar. Figure 1–7 is a composite summing of both the posterior ninth nerve and chorda tympani (CT) responses together with corresponding typical preference-aversion functions of the two-bottle Richter test. Sweet sensitivity can be better studied in other species like dog, hamster, squirrel, guinea pig or monkey. We chose the squirrel monkey whose chorda tympani shows the good response to sugar seen in Figure 1–8 with a clear seriation of sugar effectiveness as seen in Figure 1–9. Fructose arouses the greatest chorda response followed by sucrose, then glucose and other sugars each of which are not significantly different

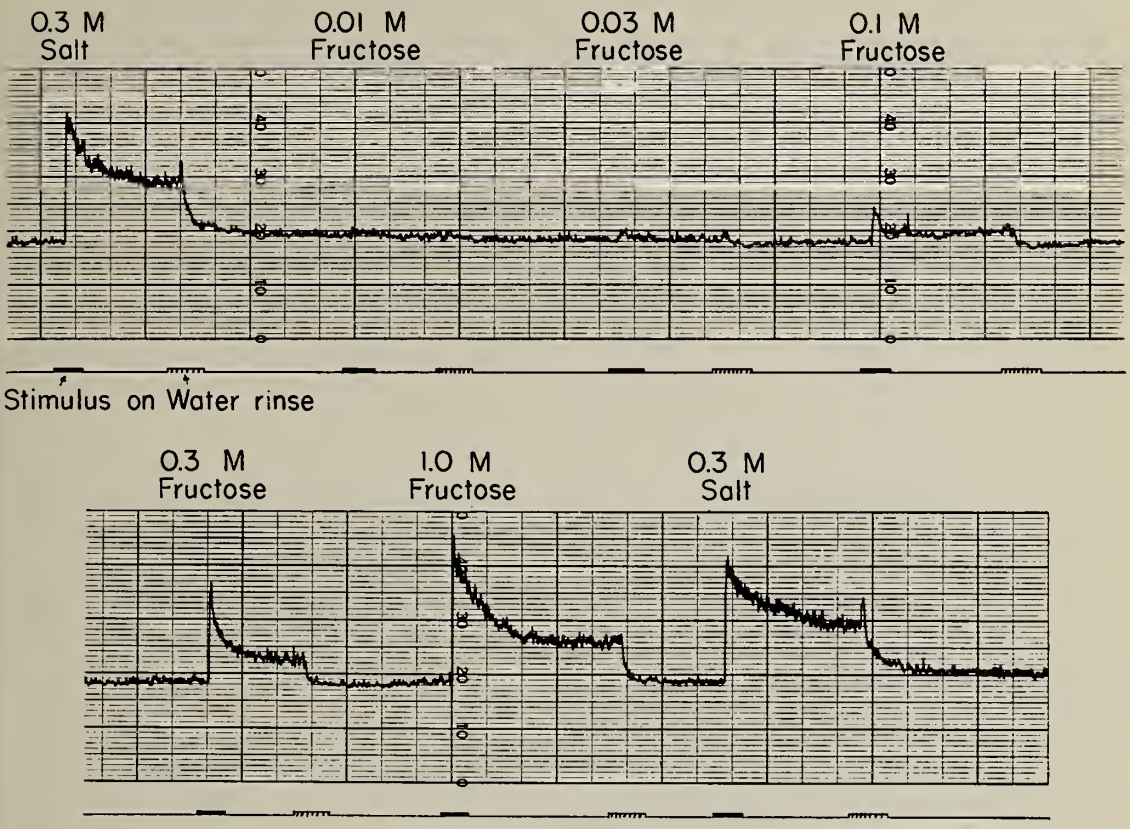


Figure 1–8. Summated electrophysiological responses of squirrel monkey chorda tympani to a test salt and an ascending concentration series of fructose solutions. Time scale: one div. = 20 secs. (Reproduced with permission; Pfaffmann [F].)

from each other. For purposes of comparative tests we concentrated on the relation among three: fructose > sucrose > glucose. The varying sweetness of different sugars is well documented for man [7,26] as are their behavioral effectiveness in animals and man.[30,9] A similar electrophysiological seriation would be expected (see also Andersen, et al.[1]; Diamant, et al.[11]).

Figure 1–10 shows two-bottle preference tests of one hour duration. Water intake is not plotted because it is essentially zero. We discovered that squirrel monkeys were so avid for any one of these sugars that in an ordinary ad lib twenty-four hour test, an animal almost drowned itself in sweet fluid. Behavior became erratic, and the animal's health deteriorated, so we had to limit them by scheduled removal of water and controlled eating. A measured amount of food was available for 12 hours following the test, followed by a period of eleven hours deprivation before the next test. Then the data became very regular. The seriation on the ascending limb of the preference curves shows the order: sucrose, fructose, then glucose.

Ganchrow and Fisher [14] in my laboratory at Brown compared the effec-

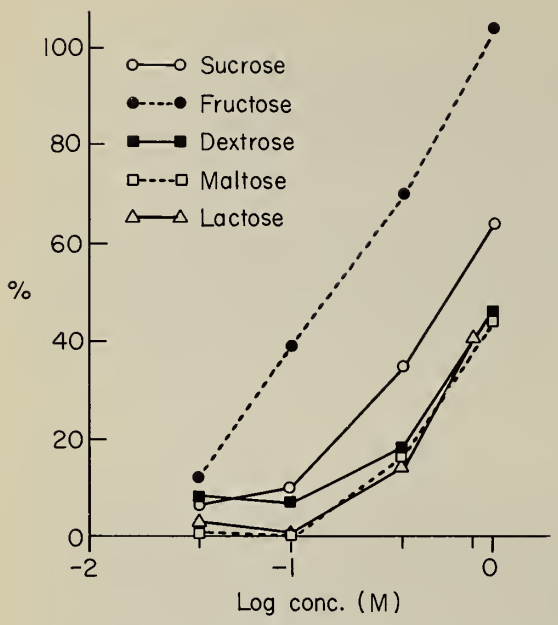


Figure 1–9. Relative chorda tympani responses of squirrel monkey to different sugars. (Reproduced with permission; Pfaffmann [F].)

tiveness of the three sugars, fructose, sucrose and glucose, for the squirrel monkey in a bar pressing task reinforced by the brief presentation of solutions of one of these sugars. Figure 1–11 shows the relative bar press-

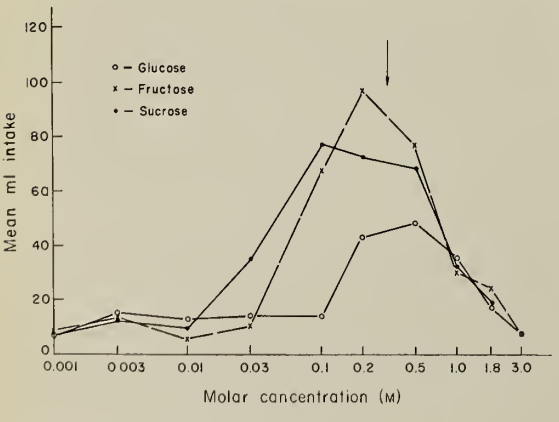


Figure 1–10. One-hour, two-bottle preference tests of squirrel monkey for three sugars. Arrow marks isosmotic point. (Reproduced with permission, Pfaffmann [G].)

ing functions across sugars. Sucrose, fructose, glucose and lactose fall off in that order. The functions from a single animal are quite regular and are in direct conflict with those seen electrophysiologically. Until this finding we had previously found a reasonably good correspondence between taste electrophysiology and behavior. These paradoxical findings, as a matter of fact, turned out to be important. This lack of correlation is not a trivial detail for ultimately it clarified for us the fundamental issue of the gustatory code for sweetness. Even with one difference in methodology, data collected at Rockefeller with the help of Bruce

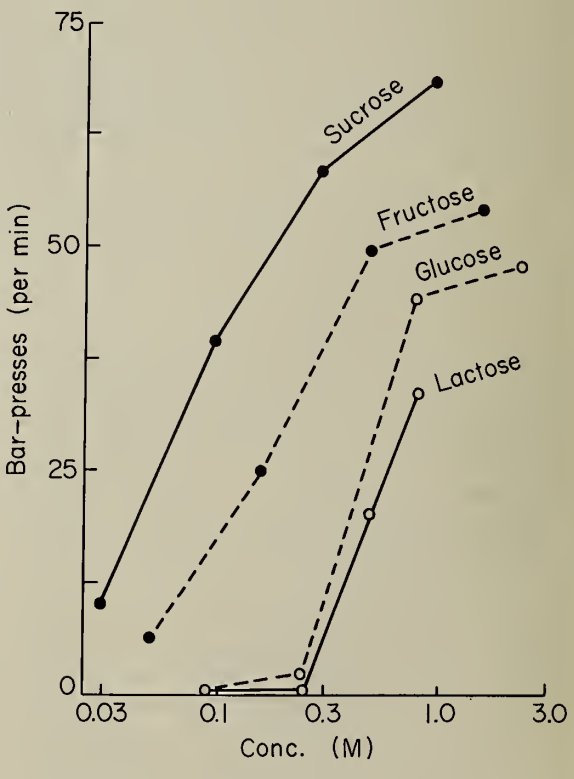


Figure 1–11. Bar pressing for different sugars as a function of concentration (Ganchrow & Fisher [14]). (Reproduced with permission, Pfaffmann [G].)

McCutcheon and Joan Colsey confirm these relations. In the Brown study, animals were trained initially on sucrose. The decrement to fructose and other sugars might have resulted from the change in substance. In our study, animals were trained initially on fructose. The training sugar did not affect the behavioral superiority of sucrose. Furthermore at maximal and equal licking rates on both sugars, i.e., with equal consummatory behavior, there is a bar press difference in favor of sucrose. Thus, when equated for consummatory behavior, sucrose retains its advantage as seen in Figure 1–12. At lower concentrations differences in licking appear and both the lick and bar press rates for sucrose exceeded the rates observed for fructose.

Figure 1–13 illustrates a slightly more sophisticated method of comparing sugars. We presented glucose and fructose in ascending and descending concentration series against a standard sucrose solution in a two-bottle balanced position test. No water was available during the one-hour test on the above schedule. The animal samples the two tubes and takes relatively greater amounts of the test solution (fructose or glucose depending on the test) with increasing concentration. At some concentration of test solution, the choice of test and standard becomes equal, marking the 50% point of the test intake curve. A sucrose concentration of 0.1 M is matched by a higher fructose concentration of 0.14 M and a still higher glucose concentration of 0.4 M. For equal palatability sucrose > fructose > glucose in terms of molar concentration.

I put together lick rate and preference intake data in Figure 1–14. Norman Guttman [17] originally showed that, contrary to preference, rate of bar pressing didn't show a peak performance and then drop off as a function of sugar concentration. Rather he found ascending functions to a plateau. The difference between intake preference and operant non-peaked curves was enhanced by intermittent schedules of reinforcement. We theorized that the reduced intake on the intermittent schedule would reduce post ingestive factors such as hyperosmolarity or negative sensory input from the gut. That may be a debatable interpretation, but in general it is rather helpful to think of two factors: one driving the behavior forward and the other turning it off. Small volume intakes, as in operant schedules, yield rising functions to a plateau as a reflection of the sensory positive instigating factor.

Whereas fructose > sucrose > glucose in the total chorda tympani discharge, single fibers, as first shown by Dr. Linda Bartoshuk,[3] do not always show this order. Some fibers discharge more actively to fructose much as in the total nerve, but others respond more to sucrose than to fructose or other sugars. Because these early experiments were carried out on only a relatively small number of fibers, we wondered about a larger population. In more recent experiments at the Rockefeller University, which Dr. Frank will describe, we applied Frank's best stimulus designator to both sugar and salt fibers. Figure 1–15 shows one group of fibers with similar response patterns in which sucrose is more effective than any of the other stimuli employed, including salts, acid, and quinine. In the

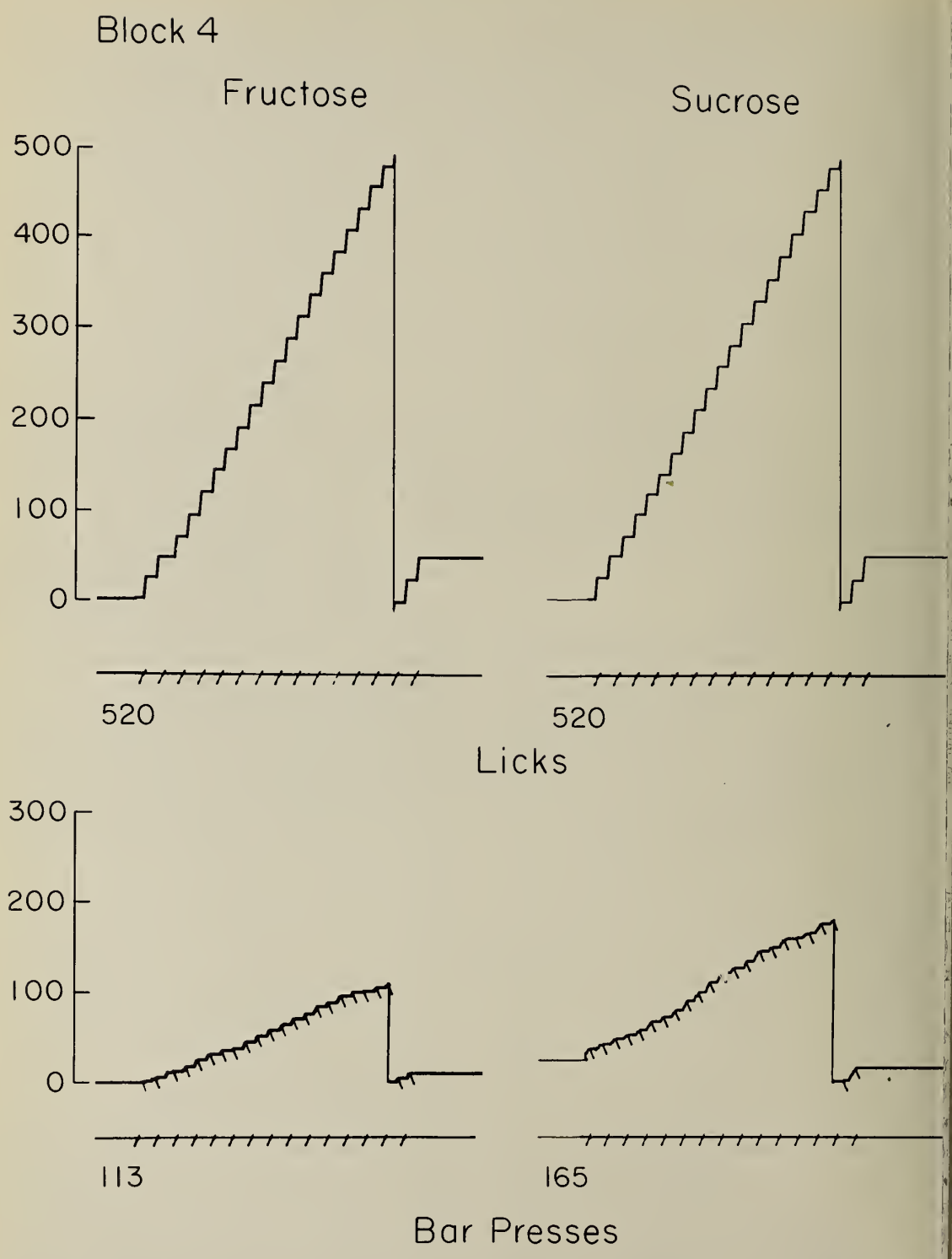


Figure 1–12. Cumulative records of licking and bar pressing for fructose and sucrose in one animal. (Reproduced with permission, Pfaffmann [6].)

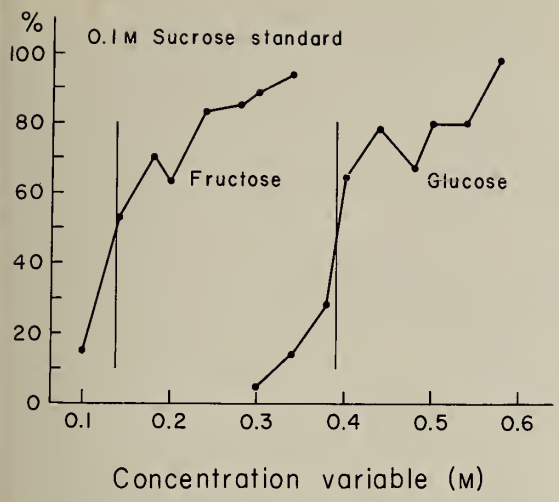


Figure 1–13. Preference for fructose and glucose relative to 0.1 M sucrose. (Reproduced with permission, Pfaffmann [G].)

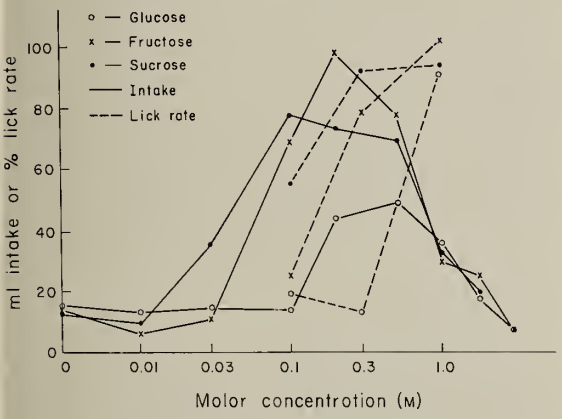


Figure 1–14. Operant lick rates for glucose, fructose, and sucrose superimposed on a plot (Figure 1–10) of the volumes of these sugars ingested by the same animals.

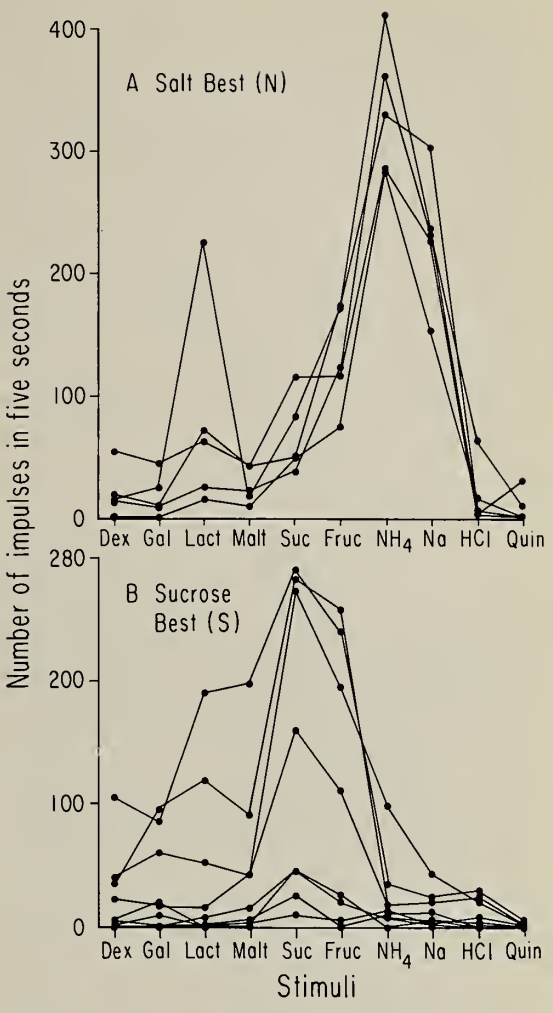


Figure 1–15. Response profiles of salt-best and sugar-best fibers of squirrel monkey. (Reproduced with permission, Pfaffmann [H].)

other group, which also responded to sugars, fructose was more effective than sucrose, but salts were much more effective, indeed these appeared to be salt-best units. If sugars only had been tested one might have labelled these simply as fructose fibers. I went back through Dr. Bartoshuk's original protocols and discovered that, indeed, good fructose elements were also strongly salt sensitive; those that were maximally responsive to sucrose showed some but not much salt responsivity. Thus there appear to be two distinct groups of sensitivity patterns to sugars.

We now believe that the sucrose-best (S fibers) signal sweetness and the salt-best (N fibers) probably signal saltiness. Why the salt fibers should also be activated by sugar especially fructose is probably a biochemical or biophysical question that must be

answered in terms of receptor properties. Whenever the sucrose fiber system is activated, the organism senses sweet; whenever the other is activated, there may be saltiness or some other more complex modality. This, of course, is close to the traditional view of basic modalities of the taste each mediated by specific labelled lines for their particular sensory quality. We think of them as clusters of labelled lines, all very similar as to the best stimulus, but not necessarily identical especially in their side bands of sensitivity.

I think we must still retain some aspects of the cross fiber patterning concept.[28,12] When one considers all the chemicals that exist to which taste responses can be made, there seem to be far too many to be coded only by a small population (four or five) of highly specific primary sensitivities. There is much sensory information in the multiple responsivity leading to different ratios of response, in short the "across fiber pattern." I would opt for *both*; a labelled line code, which is the traditional view, and an across fiber pattern code that provides modulation. There is no necessary incompatibility between these two mechanisms, indeed one may say there is complementarity.

Dr. Vincent Dethier is known for his beautiful work on the blowfly taste preparation, which for years was taken as the prime example of specific or specialized taste receptors. There are four sensory cells at the base of the chemosensory hair of the blowfly: one for sugar, one for salt, one for water, and the fourth still uncertain. Those three are highly specific when tested with the traditional basic stimuli, but with a wider range some preparations responded to two or more stimuli. Salt receptors are found to be reactive to some of the amino acids. In eating of complex foods, the animal distinguishes behaviorally amongst these materials. Since there is no specific amino acid receptor, the only information available is by way of the across fiber information derived from the relative rates of firing among the three identified receptors. In a recent paper, Dethier [10] accepts across fiber patterning as an essential adjunct to specificity. In insects and other similar invertebrates there is "a surfeit of stimuli: a paucity of receptors." In mammals we would retain the across-fiber-pattern concept but as an adjunct of a labelled line set built around four or a few basic sensitivities. In both insects and mammals, behavioral data play an important role in leading to this interpretation. One must have some way of knowing what the electrical message we read on the oscilloscope means to the organism.

Another currently active line of investigation into the central nervous mechanisms of taste was initiated by Dr. Ralph Norgren [27] in our laboratory. The solitary tract and nucleus of the medulla is the primary input path for the taste fibers of the seventh, ninth and tenth nerves. The phantom sketch presented in Figure 1–16 shows taste inputs to the solitary tract. The posterior solitary tract receives input from the vagus as well as visceral receptors of the internal organs of the body (not shown).

The ascending afferent fibers from the solitary nucleus pass through the pons to the thalamus. The thalamocortical pathway, well delineated by Robert Benjamin and his colleagues [6]

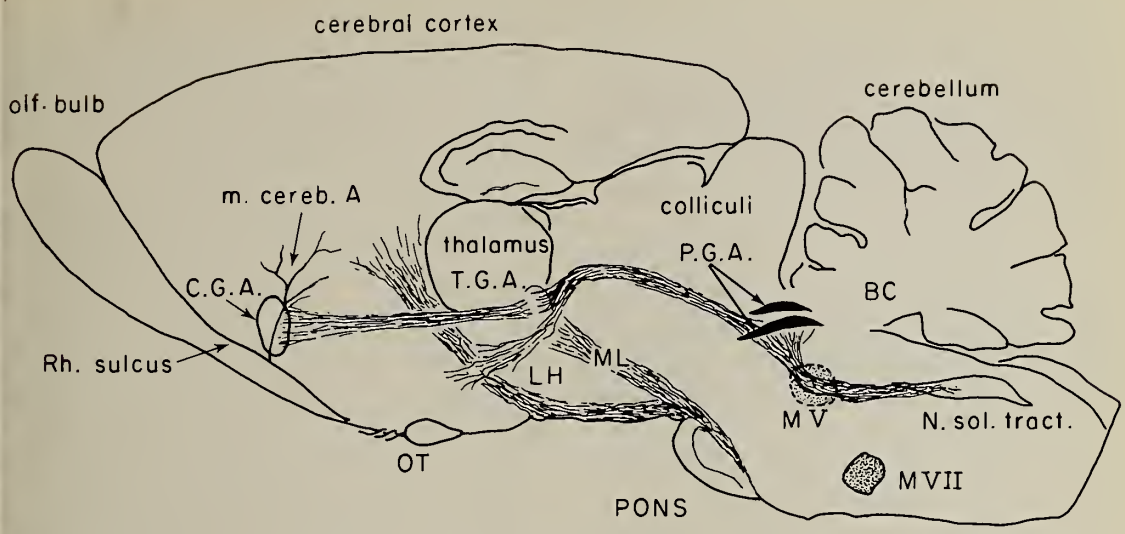


Figure 1–16. Phantom sketch of taste pathways of rat brain.

reaches the cortical gustatory area on the orbital surface of the rat brain. That system had been known for some time, but not the detailed pathway from medulla to thalamus. Previous workers had difficulty tracing the solitary nucleus projection for good reason. Until recently, it was thought that second order neurones passed directly to the thalamus. We know now there is a synaptic break in the pontine gustatory area (PGA), a nucleus of third order neurons in the brachium conjunctivum. Surrounding the taste cells are cerebellar fibers of passage of the brachium and cells and fibers of the trigeminal system as well as other systems. From the pontine taste area ascending taste fibers literally bifurcate, one branch to the thalamus and one branch ventrally to the region of the substantia innominata of the anterior ventral forebrain. During the past year, Ralph Norgren has confirmed the fact that cells in the ventral forebrain respond to gustatory stimuli and that PGA gustatory cells can be fired antidromically from electrodes in the substantia innominata. In the PGA itself, he and I have found cells that are specific to one, two, three or four of the basic taste stimuli, usually with a best stimulus much like the sensitivity at the periphery. We also see signs of convergence upon the single pontine units of distinct inputs from anterior and posterior receptive fields together with certain interactions I shall not discuss here. Lesions in substantia innominata produce aphagia and adipsia, rather like that of the hypothalamic syndrome.[16] Thus it appears this ventral pathway may be the route by which taste joins with or interacts with the neural systems controlling food and water intake.

This relation of taste to the food intake systems is important for understanding the "sweet tooth." Much behavioral evidence suggests that the preference for sugar is directly linked to hunger; when the animal is hungry, its intake of sugar is increased when satiated sugar intake is reduced. Other recent evidence on the effect

of 6-hydroxy-dopamine is pertinent here. Smith, Cooper, and Breese [33] and Marshall and Teitelbaum [25] show that intracranial injections of this agent produces chemical lesions which mimic many features of the lateral hypothalamic aphagic syndrome. Associated with the aphagia is a loss of sugar preference. Sorenson, Ellison and Masuoka [34] showed that this is not due to a general taste deficit for the aversion to quinine remains and if anything is enhanced, the loss was specific for the positively attractive sucrose. A drug which impairs appetitive behavior also impairs the response to sugar as food. All of which leads me to the conclusion that sugar preference is an aspect of hunger. We should study the sugar preference not only with regard to its unique taste properties but as a carbohydrate with nutritive and caloric value.

Figure 1–17 is a multi-electrode recording showing the relationship of the magnitude of responses from the anterior and posterior tongue upon a particular electrode in the pontine taste area. I wanted to show particularly the proximity of this to the locus coeruleus, which is one origin of the dorsal noradrenergic fiber system. When Dr. Norgren first described his lesion experiments, showing degeneration into the ventral forebrain there was concern that he might have,

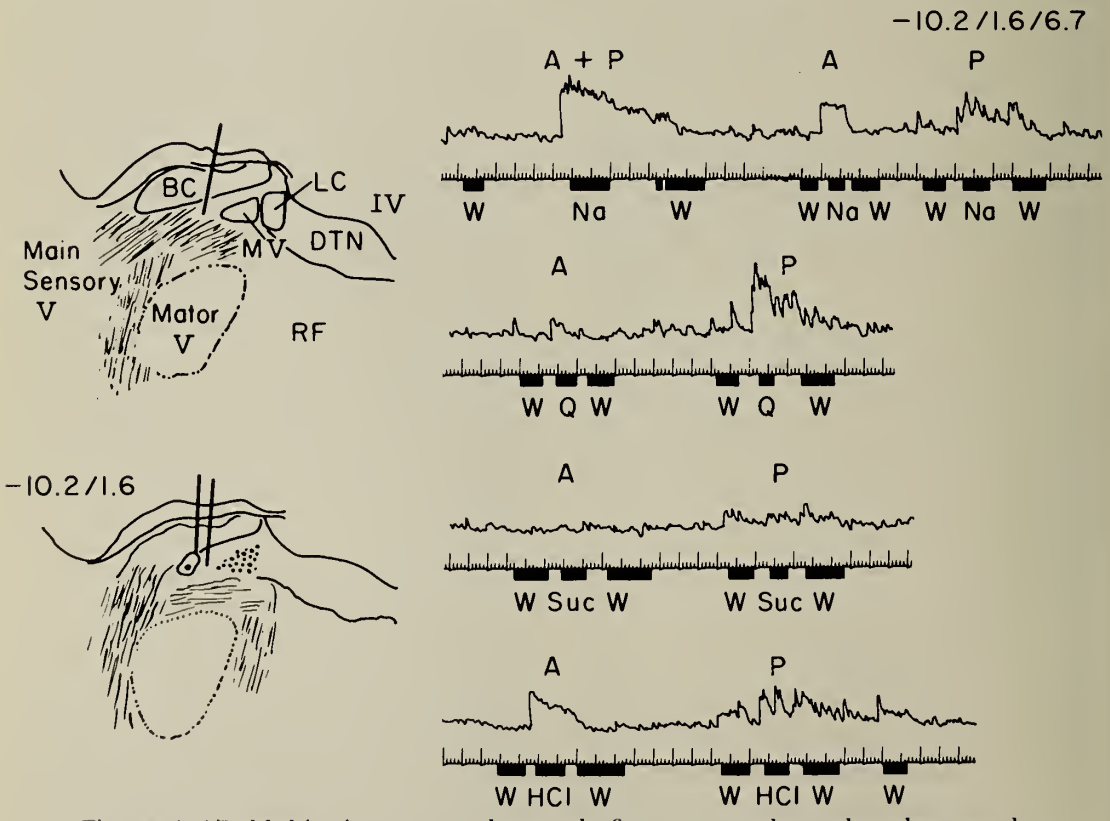


Figure 1–17. Multiunit summated records from outer electrode subsequently marked by lesion at bottom of -10.2/1.6. Abbreviations: BC=brachium conjunctivum; DTN=dorsal tegmental nucleus; LC=locus coeruleus; MV=mesencephalic trigeminal nucleus; IV=fourth ventricle; RF=reticular formation.

in fact, interfered with the noradrenergic ascending system. The effects of 6-hydroxy-dopamine I have been describing are not related to the involvement of the locus coeruleus in Norgren's experiments because all his lesions clearly avoided that structure. The fact that it appears in this brain section is purely coincidental. The pontine taste area is a small dense nuclear area of small cells where it is difficult to isolate cells for microelectrode recording, but we can pick them up around the fringes where the cells are less densely packed and intermingled with other fiber tracts and systems. We haven't any evidence on this yet, but one might expect to see the beginnings of specialization of the gustatory input at the pontine bifurcation. Discriminatory information going to the thalamus and cortex may have different properties than that projecting ventrally into the substantia innominata. The latter incidentally is the end point of a fairly massive olfactory input to the ventral forebrain so that taste and smell come together in an area demonstrated to have an important role in feeding behavior.

Of particular interest are the phylogenetic relationships. Figure 1–18 shows the wide distribution of taste buds over the external body surface of the bottom feeding catfish. In this species taste can function as an exteroceptor, highly adapted to the detection of nutrient chemicals in its fluid environment. These fish also possess taste buds in the more usual locations, the oral cavity and palate, their only locus in most other species. Paralleling this great development in number and location of taste receptors is a great enlargement of the gustatory apparatus of the brain. Figure 1–19 is a schematization of the brain of the catfish. C.J. Herrick studied the brain and behavior of the catfish extensively.[18] In fact, this figure is a tracing of part of his diagram of the seventh cranial nerve input. The VIIth nerve sensory branch supplies the taste buds of the barbels, lips and external body surface. The taste buds of the mouth and palate of the catfish are innervated by the vagus (Xth) cranial nerve (not shown in the figure). The figure shows the sensory fibers of the VIIth nerve entering the medulla where there is the first gustatory nucleus corresponding to the first gustatory relay of the nucleus solitarius of mammals. The nerve supply is so rich that the facial lobe appears as a distinct enlargement on the dorsal medulla of the catfish. From here the fibers stream up to the secondary gustatory nucleus located near the anterior cerebellum whose fibers stream through and around the nucleus in a

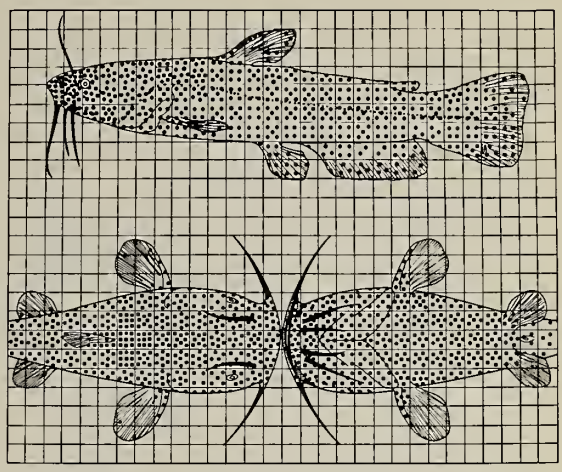


Figure 1–18. Distribution of taste buds on skin of catfish. Each block dot represents 100 taste buds. Higher concentration on barbels indicated by solid black. (Reproduced with permission, Atema [2].)

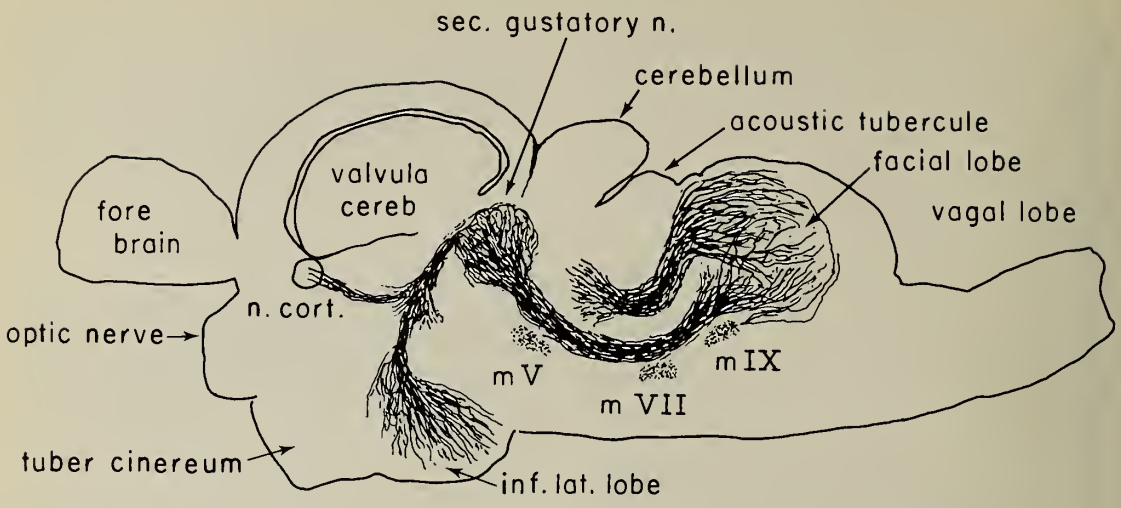


Figure 1–19. Partial tracing of VIIth nerve taste pathways of fish brain. (Reproduced from Herrick [18].)

manner resembling that of the pontine taste area of the rat. In the fish this is a relatively large structure compared with its small size in the rat. The third order fibers arise from the secondary gustatory nucleus and distribute largely to the ventral lateral inferior lobe, the analogue of the mammalian hypothalamus. Herrick was very interested in the behavioral control of feeding exerted by this system. He traced a series of connections back from the ventral inferior lateral lobe to the brain stem involved in feeding reflexes.

Atema [2] carried out a very interesting experiment. In one group of catfish, he selectively ablated the facial lobe, the central nucleus of the VIIth nerve innervating most of the body taste receptors. In another he cauterized the vagal lobes to which taste buds of the mouth cavity project. After recovery the fish was returned to the aquarium. Normally the catfish is very adept at finding all kinds of delicious decaying products in the somewhat slowly moving streams or bodies of water it inhabits. Initially, the facial lobe operated fish couldn't find food, but could swallow it when placed in its mouth. Upon removing the vagal lobe the animal could find food but didn't swallow. The behavioral distinctions are very interesting because it suggests that each nerve and its receptive field controls different clusters of feeding reflexes.

Macht [24] found that a chronic decerebrate cat couldn't feed itself nor select nutrients but would swallow when food was placed in the mouth. Meat which animals normally swallow would be rejected when dipped in bitter solution. A sugar impregnated pledget of cotton would be accepted. Thus the decerebrate mammal has nearly all the reflexes for ingestion organized at the brain stem level. We know there are taste systems above this brain level and one might well ask what additional functions they add. If one bypasses the afferent apparatus entirely by means of a chronic stomach tube there is no preference-aversion in the rat.[5] Deficits in taste

thresholds can be produced by lesions at various levels of the central taste system. In the brain stem losses are permanent. But lesions higher in the system, in thalamus and especially the cortex, show less severe impairment and recovery of function is often striking. The severity of induced deficit in taste guided preference and/or discrimination seems to indicate a hierarchy of neural mechanism extending from medulla, through thalamus to cortex. This is matched by indications from studies of the recoverability of function.[6] The more cephalad levels of the nervous system must be involved in searching out, seeking and selecting food, in controlling instrumental behaviors to bring the tastant to the sensory fields of the hind brain, where more automatic ingestive responses are organized. The more cephalad components mediate adaptability and learned modifications.

Conditioned taste aversion [15] is the most recently described and most dramatic example of how an evolutionarily derived preference, as for sweet, can be reversed to the point that sweetness will be avoided, and aversion replaces avidity. That is an extreme instance of Professor LeMagnen's [23] earlier demonstrations that preferences for nutrients and sugar can be increased or decreased by their nutritional consequences. Paul Rozin [31] has admirably documented this effect in learned specific hungers. This post-ingestive effect, both metabolic and sensory in the gastric and lower digestive system, not merely modulates preference behavior but in fact can alter or reverse what "seems" good to the organism. The taste apparatus is preset or pretuned probably genetically for the neonate when it comes into the world for animal or man. But attached to or included in this fundamental pattern of acceptance and/or rejection mechanisms is the potential for modification by a "learning like" adaptation. I say "learning like" only to distinguish taste learning from more familiar associative learning and conditioning which differs in a number of interesting parameters. Perhaps these newly described taste pathways in mammals in the ventral forebrain via the pontine taste nucleus play a particular role in such influences. All this, of course, opens up another very exciting prospect for future research.

REFERENCES

1. Andersen, H.T., Funakoshi, M., and Zotterman, Y.: Electrophysiological investigation of the gustatory effect of various biological sugars. *Acta Physiol. Scand.*, 56: 362–375, 1962.
2. Atema, J.: Structures and functions of the sense of taste in the catfish. *Brain, Behav. Evol.*, 4: 273–294, 1971.
3. Bartoshuk, L.: Reported in Pfaffmann, C. Taste preference and reinforcement. In Tapp. J. (Ed.), *Reinforcement and Behavior*, New York, Academic Press, 1969.
4. Bicchieri, M.G. (Ed.), *Hunters and Gatherers Today,* New York, Holt, Rinehart and Winston, 1972.
5. Borer, K.: Disappearance of preferences and aversions for sapid solutions in rats ingesting untasted fluids. *J. Comp. Physiol. Psychol.*, 65: 213–221, 1968.

6. Burton, H., and Benjamin, R. M.: Central projections of the gustatory system. In Beidler, L. M. (Ed.), *Handbook of Sensory Physiology, Vol. IV*, Berlin, Springer Verlag, 1971, pp. 148–164.
7. Cameron, A.T.: The taste sense and the relative sweetness of sugars and other sweet substances. *Scientific Report Series No. 9*, New York, Sugar Research Foundation, 1947.
8. Deerr, N.: *History of Sugar, Vols. 1 and 2,* London, Chapman Hall, 1949.
9. Desor, J.A., Maller, O., and Turner, R.E.: Taste in acceptance of sugars by human infants. *J. Comp. Physiol. Psychol.*, 84: 496–501, 1973.
10. Dethier, V.G.: A surfeit of stimuli: A paucity of receptors. *Amer. Scient.*, 59: 706–715, 1971.
11. Diamant, H., Funakoshi, M., Strom, L., and Zotterman, Y.: Electrophysiological studies of human taste nerves. In Zotterman, Y. (Ed.), *Olfaction and Taste,* Oxford, Pergamon Press, 1963, pp. 193–203.
12. Erickson, R.P.: Sensory neural patterns and gustation. In Zotterman, Y. (Ed.), *Olfaction and Taste,* Oxford, Pergamon Press, 1963, pp. 205–213.
13. Frings, H.: Biological backgrounds of the "Sweet Tooth." *Turtox News,* 24, No. 8, 1946.
14. Ganchrow, J., and Fisher, G.L.: Two behavioral measures of the squirrel monkey's (*Saimiri Sciureus*) taste for four concentrations of five sugars. *Psychol. Rep.*, 22: 503–511, 1968.
15. Garcia, J., Hankins, W.G., and Rusiniak, K.W.: Behavioral regulation of the milieu interne in man and rat. *Science,* 185: 824–831, 1974.
16. Gold, R.M.: Aphagia and adipsia following unilateral and bilaterally asymmetrical lesions in rats. *Physiol. Beh.*, 2: 211–220, 1967.
17. Guttman, N.: Operant conditioning, extinction and periodic reinforcement in relation to concentration of sucrose used as a reinforcing agent. *J. Exp. Psychol.*, 46: 213–224, 1953.
18. Herrick, C.J.: The central gustatory paths in the brains of bony fishes. *J. Comp. Neurol. Psychol.*, 15: 375–456, 1905.
19. Jacobs, H.L.: The osmotic postingestion factor in the regulation of glucose appetite. In Kare, M.R. and Halpern, B.P. (Eds.), *The Physiological and Behavioral Aspects of Taste,* Chicago, U. of Chicago Press, 1961, p. 31.
20. Jolly, A.: *The Evolution of Primate Behavior,* New York, Macmillan Co., 1972.
21. Kare, M.: Comparative study of taste. In Beidler, L.M. (Ed.), *Handbook of Sensory Physiology, Vol. IV,* Berlin, Springer Verlag, 1971, 278–292.
22. Kolata, G.B.: !Kung Hunter-Gatherers: Feminism, diet and birth control. *Science*, 185: 932–934, 1974.
23. LeMagnen, J.: Habits and food intake. In Code, C.F. (Ed.), *Handbook of Physiology,* Section 6: *Alimentary Canal, Vol. 1, Control of Food and Water Intake,* Washington, American Physilogical Society, 1967, pp. 11–30.
24. Macht, M.B.: Subcortical localization of certain "taste" responses in the cat. *Fed. Proc.*, 10: 88, 1951.
25. Marshall, J.F., and Teitelbaum, P.: A comparison of the eating in response to hypothermic and glucoprivic challenges after nigral 6-hydroxydopamine and lateral hypothalamic electrolytic lesions in rats. *Brain Res.*, 55: 229–233, 1973.
26. Moskowitz, H.R.: The sweetness and pleasantness of sugars. *Amer. J. Psychol.*, 84: 387–405, 1971.
27. Norgren, R., and Leonard C.: Taste pathways in rat brainstem. *Science,* 173: 1136–1139, 1971.
28. Pfaffmann, C.: The afferent code for sensory quality. *Amer. Psychol.*, 14: 226–232, 1959.
29. Pfaffmann, C., Fisher, G.L., and Frank, M.: Sensory and behavioral factors in taste preference. In Hayaski, T. (Ed.), *Olfaction and Taste II,* Oxford, Pergamon Press, 1967, pp. 361–381.
30. Richter, C.P., and Campbell, K.H.: Taste thresholds and taste preferences of rats for five common sugars. *J. Nutrition, 20*: 31–46, 1940.
31. Rozin, P.: Thiamine specific hunger. In Code, C.F. (Ed.), *Handbook of Physiology,* Section 6: *Alimentary Canal, Vol. 1, Control of Food and Water Intake,* Washington, American Physiological Society, 1967, pp. 411–431.

32. Scott, J.W., and Pfaffmann, C.: Olfactory input to hypothalamus: Electrophysiological evidence. *Science,* 158: 1592–1594, 1967.
33. Smith, R.D., Cooper, B.R., and Breese, G.R.: Growth and behavioral changes in developing rats treated intracisternally with 6-hydroxydopamine: Evidence for involvement of brain dopamine. *J. Pharm. Exp. Therap.,* 185: 609–619, 1973.
34. Sorenson, C.A., Ellison, G.D., and Masuoka, D.: Changes in fluid intake suggesting depressed appetite in rats following central catecholaminergic lesions. *Nature new Biol.,* 237: 279–281, 1972.
35. Tindale, N.B.: The Pitjandjara. In Bicchieri, M.G. (Ed.), *Hunters and Gatherers Today,* New York, Holt, Rinehart and Winston, 1972, pp. 217–268.
36. Warren, R.M. and Pfaffmann, C.: Suppression of sweet sensitivity by potassium gymnemate. *J. Appl. Physiol.,* 14: 40–42, 1959.

Additional References (Cited in Figure Legends)

A. Beidler, L.M.: Innervation of rat fungiform papilla. In Pfaffmann, C. (Ed.), *Olfaction and Taste III,* New York, Rockefeller Press, 1969, pp. 352–369.
B. Fish, H.S., Malone, P.D., and Richter, C.P.: The anatomy of the tongue of the domestic Norway rat: I. The skin of the tongue; the various papillae; their number and distribution. *Anat. Rec.,* 89: 429–440, 1944.
C. Halpern, B.P., Bernard, R.A., and Kare, M.R.: Amino acids as gustatory stimuli in the rat. *J. Gen. Physiol.,* 45: 681–701, 1962.
D. Pfaffmann, C.: Taste preference and aversion following lingual denervation. *J. Comp. Physiol, Psychol.,* 45: 393–400, 1952.
E. Pfaffmann, C.: Taste mechanisms in preference behavior. *Amer. J. Clin. Nutrition,* 5: 142–147, 1957.
F. Pfaffmann, C.: Taste preference and reinforcement. In Tapp, J. (Ed.), *Reinforcement and Behavior,* New York, Academic Press, 1969.
G. Pfaffmann, C.: Physiological and behavioural processes of the sense of taste. In Wolstenholme, G.E. and Knight, J. (Eds.), *Taste and Smell in Vertebrates,* A Ciba Foundation Symposium, London, Churchill, 1970, pp. 31–50.
H. Pfaffmann, C., Frank, M., Bartoshuk, L. and Snell, T. Coding gustatory information in the squirrel monkey chorda tympani. *Progress in Psychobiology and Physiological Psychology, Vol. 6,* New York, Academic Press, 1976, pp. 1–24.

DISCUSSION

Le Magnen: I have a comment about this. The relationships among brain sites are of great importance for the concepts of energy balance and sweet preference.

Pfaffmann: In mentioning the interaction of taste and the projections of the taste system, I am well aware that I didn't go into the evidence for other pathways. There are other connections with the olfactory system. Indeed, John Scott and I [32] described a pathway directly from the olfactory lobe to the lateral hypothalamus and that also bifurcated to the dorsal medial thalamus. The relationship among these other pathways is certainly an important unanswered question for further investigation.

Jacobs: I'd like to comment briefly on your suggestion that hyperosmolality might be one of the shut-off signals inhibiting intake of concentrated sucrose solutions. This was originally

suggested by the early work of McCleary [1] and Shuford.[2] I have discussed this literature in detail previously,[3,4] showing that hypertonic sugar solutions *do not* act like isosmotic NaCl solutions as required by the hyperosmolality theory. An example of this failure is provided by LeMagnen's paper in this symposium (p. 358, Fig. 25–2) when he showed that hypertonic NaCl loads increase water intake and decrease NaCl intake, but *fail* to decrease hypertonic sucrose intake. The critical experiment on this issue was completed by Yin and Brobeck,[5,6] who showed that intravenous hypertonic glucose loads produced only minor and transient changes in serum osmolality in comparison to isosmotic hypertonic NaCl loads. These findings are consistent with the behavioral observation [3] that unlike NaCl, a known dehydrator, hypertonic glucose loads *fail* to produce thirst.

PFAFFMANN: I am glad to have these comments and references.

STEINER: You mention the vagal input to the solitary nucleus area. Because of this vagal input you would think that even at the level of medulla there could be interactions with visceral sensations and even possibly sweet or sugar information from the pancreas.

The other remark is related to the observations on birds. I don't believe that all birds should be collected under the same umbrella. Birds differ anatomically, physiologically and behaviorally. Different types of birds may behave differently with respect to sweet preference.

PFAFFMANN: A while ago I thought I was on the track of an interesting group of birds called honey gliders, but as I read more about them, it turned out they are not so much after the honey, as the wax and dead bees and other insects in the honey comb. Unfortunately, a "sweet tooth" for honey seemed not to be important in this case.

But hummingbirds have a "sweet tooth." Most nectars are composed of sucrose, glucose and/or fructose equivalent to 0.24–2.10 M. In taste tests hummingbirds detect differences in concentration. But they cannot survive on sugar alone; the mixture of sugar plus insects also attracted by the nectar provides the additional material for balanced nutrition.[7]

RODIN: Let me comment on Steiner's first point. Powley's [8] recent work shows that the hypothalamic hyperphagic syndrome can be reversed in lesioned rats by severing the vagus. Overeating then stops and the animal returns to normal weight.

STEINER: Considering that different

[1] McCleary, R.A.: Taste and post-ingestion factors in specific hunger behavior. *J. Comp. Physiol. Psych.* 46:411, 1953.

[2] Shuford, E.: Palatability and osmotic pressure of glucose and sucrose solutions as determinants of intake. *J Comp. Physiol. Psych.* 52:150, 1959.

[3] Jacobs, H.L.: The osmotic postingestion factor in the regulation of glucose appetite. In Kare, M. and Halpern, B. (Eds.). *The Physiological and Behavioral Aspects of Taste,* Chicago, Univ. of Chicago Press, 1961, pp. 16–28.

[4] Jacobs, H.L.: The interaction of hunger and thirst: Experimental separation of osmotic and oral-gastric factors in the regulation of caloric intake. In Wayner, M. (Ed.). *Thirst,* Oxford, Pergamon Press, 1964, pp. 117–137.

[5] Yin, T.H., Hamilton, C.L. and Brobeck, J.R.: Effect of body fluid disturbances on feeding in the rat. *Amer. J. Physiol.* 218:1054–1059, 1970.

[6] Yin, T.H., Hamilton, C.L. and Brobeck, J.R.: Food intake of rats given hypertonic solutions by gavage and water intravenously. *Proc. Soc. Exp. Biol. and Med.* 133:83–85, 1970.

[7] Pfaffmann, C.: Phylogenetic origins of sweet sensitivity. In *Olfaction and Taste V.* Academic Press, 1975.

[8] Powley, T.L. and Opsahl, C.A.: Ventromedial hypothalamic obesity abolished by subdiaphragmatic vagotomy. *Amer. J. Physiol.* 226: 25–36, 1974.

types of sensory messages related to food (gustatory, olfactory, tactile, hunger, etc.) might reach the hypothalamic structures and from this level activate motor and secretory processes one might suggest that mastication, salivation, or the complex of "consumatory behavior" be viewed as processes, not necessarily regulated by cortical structures. It is obvious, that cortical influences can participate in these processes and modulate them but the primary regulation is subcortical.

COLLIER: This question stems from a bias of mine. I have always been bothered by the fact that most preference experiments examine only carbohydrate intake. An animal also has the problem of protein and fat to solve. One could invent an asymmetrical model proposing that animals eat protein and fats only when they are satiated for carbohydrates. It would assume a taste system responsive to carbohydrates which you have shown here and no specific system for protein and for fat. Such an asymmetrical model is implied by the lack of interest most investigators seem to have in the intake of other dietary components when they are studying sugar intake. It seems to me, however, that you can not assume that protein, fat, and sucrose requirements do not interact and that all three along with other dietary requirements are represented in your primary sensory system. Thus sucrose intake must be . . . should be, a function of the state of the organism with respect to other nutrients. Contrary to mothers and dietitians, (rapidly growing) baby rats select the highest proportion of protein in the diet compared to all other age rats.

PFAFFMANN: There is partial evidence on that, I think. You are quite right in implying that the taste investigators have neglected the rest of the natural taste spectrum. But I think there is pretty good evidence that the proteins are not terribly good taste stimuli, except for a few recently discovered sweet proteins. There is a question of whether the breakdown products of the amino acids or even polypeptides are involved. Generally the amino acids are sweet. As a class, amino acids didn't seem to stimulate a special system. They ended up stimulating primarily the classical modalities and that makes protein self-selection such a puzzle. As far as the fatty acids or anything in the lipid side, we just don't know.

RICHTER: I was a bit bothered, as was Dr. Collier, that Dr. Pfaffmann didn't mention in his discussion of the evolution of the sweet taste anything about fats. I bring this up because in our experiments we found many instances in which there is an inverse relationship between sweet taste and the appetite for fats. There are many instances. One of the best is seen in the pancreatectomized animal which ordinarily shows a very marked increase in appetite for sugar, but if you give the animal access to olive oil at the same time, the sugar intake decreases completely. Many birds do have a high appetite for fat. That's why you give them suet. It can be that on the high fat diets, they just don't eat sugar. In this whole discussion of sweet taste, we have to take into account appetite for fats and also proteins, but you can't just leave it there. You have to discuss the whole organism, the whole dietary regime.

PFAFFMANN: I quite agree that in the

total economy of the organism, other interactions occur in the search for calories. My main point was not that these do not occur but that in many instances we can point to sweetness as an identified sensory influence and potent initiator of acceptance behavior. Clarifying the whole realm of feedback interactions of visceral and metabolic status with the sense of taste is an exciting prospect opened to us by the discovery of the ventral taste pathway originating in the pons.

Chapter 2

THE DISTINCTIVENESS OF RESPONSES TO SWEET IN THE CHORDA TYMPANI NERVE

Marion Frank

The view of the specifictiy with which mammalian taste nerve fibers respond has been changing. In the past, the broadness of the tuning of peripheral neural elements was emphasized and it seemed as if nearly every fiber would respond to every test stimulus. Now, however, it appears that there is much more selectivity. Sensitivities often occur together in clusters and the occurrence of one sensitivity allows prediction of the absence or presence of others. Many fibers show a considerable differential sensitivity to the four test stimuli which represent the taste qualities of man. Thus, a fiber's response to a salty stimulus may be twenty- or even fifty fold greater than its response to a sour, bitter, or sweet stimulus. In fact, it proves enlightening to group fibers together on the basis of which of the taste qualities elicits the largest response rather than considering each fiber individually.

The change of view was largely due to an accumulation of observations. Electrophysiological recordings of many single taste fiber responses to more stimuli gave a fuller picture of the distribution of their sensitivities. Some similarities and differences in the chemicals various species behaviorally group together on the basis of evoked taste quality were also determined. But it was the characteristics of the sweet taste which proved to be most influential. There are mammalian taste fibers which respond very selectively to sweet solutions. Sweet is a most distinct taste quality and sweet sensitivities prove to be more distinctly represented in chorda tympani taste fiber responses than salty, sour, or bitter sensitivities.

Species differences in sensitivity to sweet

The chorda tympani nerve innervates taste receptors on the front of the tongue where man's greatest sensitivity to sweet substances resides. However, the rat chorda, on which earlier studies of single taste nerve fibers focussed, is not particularly sensitive to sweet. For instance, the summated response of this whole nerve to sucrose is small and its threshold is high. The response to 1.0 M sucrose is one-fifth the size of the response to .05 M sodium chloride and the su-

Acknowledgment: This work was supported by the National Science Foundation Grant BMS 70–00854–A04 to C. Pfaffmann.

crose threshold concentration is greater than 0.1 M.[9] The rat chorda is highly sensitive to salts.

There are other species whose chorda tympani nerves show a much greater sweet sensitivity. Two of these are the hamster and squirrel monkey. Where the rat chorda response to 0.1 M sucrose is less than 20 percent of the size of its response to equimolar sodium chloride; [9] in the hamster the sucrose response is 50 percent [3] and in the squirrel monkey it is 40 percent [7] of the salt response.

Along with the slight whole nerve response to sweet, there is also a dearth of single nerve fibers which respond vigorously to sweet substances in the rat chorda tympani. Where half of a sample of 50 rat chorda fibers respond strongly to salt (0.1 M NaCl), and nearly all respond somewhat to salt; only 15 percent respond well to sucrose (0.5 M), and half respond at all to sucrose.[5] In the hamster, on the other hand, 65 percent of a sample of 27 fibers respond to salt, only 30 percent respond strongly; but nearly 80 percent of the fibers respond to sucrose and 45 percent respond strongly.[5]

Classic view of taste nerve fiber specificity

It is evident from the response percentages just noted that many taste nerve fibers respond to more than one taste quality. In fact, classical observation is that single mammalian taste fibers are sensitive to chemicals which taste very different to man. Although some fibers respond to stimuli of just one, most respond to stimuli of two, three, or all four taste qualities. For instance, 82 percent of a sample of 76 rat and hamster chorda tympani nerve fibers responded to more than one taste quality.[5] That is, single mammalian taste fibers typically are not specific for solutions which are of just one of the taste qualities (sweet, salty, sour, and bitter) established for man.

Although they are non-specific, individual taste fibers do not all have similar response profiles; and most of them do not have profiles which mimic the sensitivity profile of the whole nerve of the species. For instance, one fiber will respond just to salts and acids, another just to sugars and salts, but another to salts, acids, and bitter stimuli. So, instead of a few types of specific fibers: one mediating sweet, one salty, one sour, and one bitter; there appear to be many types of relatively non-specific fibers, each with its own individual response profile. Thus, the classical view is that single mammalian taste fibers are broadly tuned across quality and their response profiles do not fall into a small number of simple classes with identical characteristics.[6,1]

Choice of test stimuli

Four stimuli have been used for much of the testing of single taste fiber sensitivities. They have been one intensity each of sucrose, sodium chloride, hydrochloric acid, and quinine hydrochloride. The chosen intensities of these compounds have often been very strong, at or near the molarity evoking the maximal whole nerve response. With such intense stimuli the greatest possible number of fibers will be driven to their highest response levels. In the present work, test intensities of the common taste solutions were in the middle of

the effective concentration range bounded by threshold and maximal whole nerve response for the particular species. These less intense stimuli would be expected to have more selective effects on individual taste fibers.

A CLASSIFICATION OF TASTE FIBERS BY THEIR BEST RESPONSE

Stimulus intensity and response magnitude

Figure 2–1 is a plot of stimulus concentration versus response (number of nerve impulses in five seconds) for two single hamster chorda tympani nerve fibers to the four common tastes.[3] The encircled points are for test intensities. For effective compounds, each fiber's response increases with concentration; but some compounds are not effective even at the strongest concentrations used. Of the four test stimuli (0.1 M sucrose, .03 M sodium chloride, .003 M hydrochloric acid, and .001 M quinine hydrochloride), fiber A responds best to salt but also responds a little to acid and sugar; whereas, fiber B responds best to sucrose, also responds quite strongly to salt, but does not respond to acid. Neither fiber responds to quinine. Responses to test stimuli are used to highlight the sensitivities of a fiber and constitute its response profile.

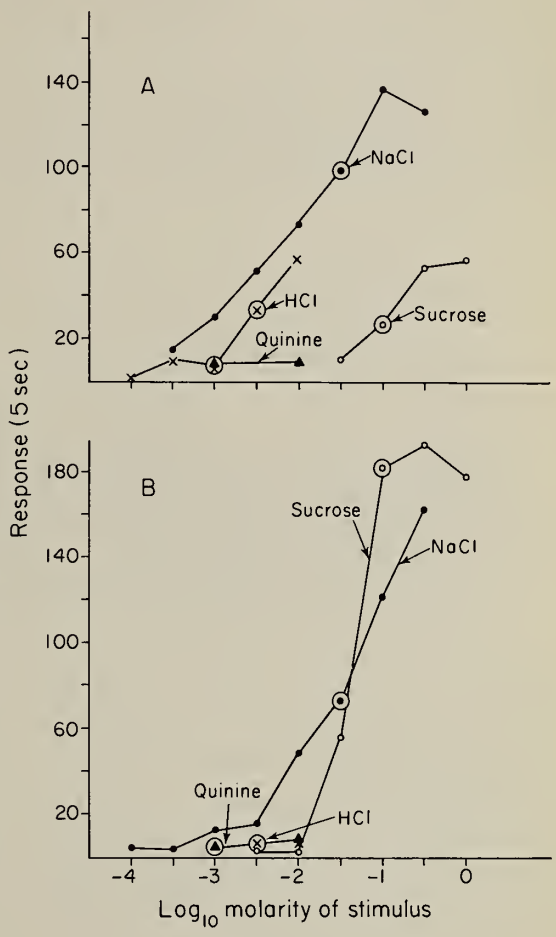


Figure 2–1. The response (number of nerve impulses in 5 sec.) of two (A and B) hamster chorda tympani nerve fibers as a function of $\log_{10}$ molarity of four tastes: sucrose, NaCl, HCl, and quinine hydrochloride. Points for responses to test molarities are encircled. (From Frank,[3] with permission of The Rockefeller University Press)

Figure 2–2 is a similar plot for two squirrel monkey chorda tympani nerve fibers.[7] In this case, however, the responses of both fibers (A and B) to the test intensities are plotted in an inset conventional response profile bar graph in which *S* represents sucrose (0.5 M); *N*, sodium chloride (0.3 M); *H*, hydrochloric acid (.01 M); and *Q*, quinine hydrochloride (.003 M). Fiber A responds very well to salt and a little to sucrose; fiber B responds very well to sucrose and a little to salt; but neither fiber responds to either acid or quinine.

Thus, it is typical for single taste nerve fibers to respond more vigor-

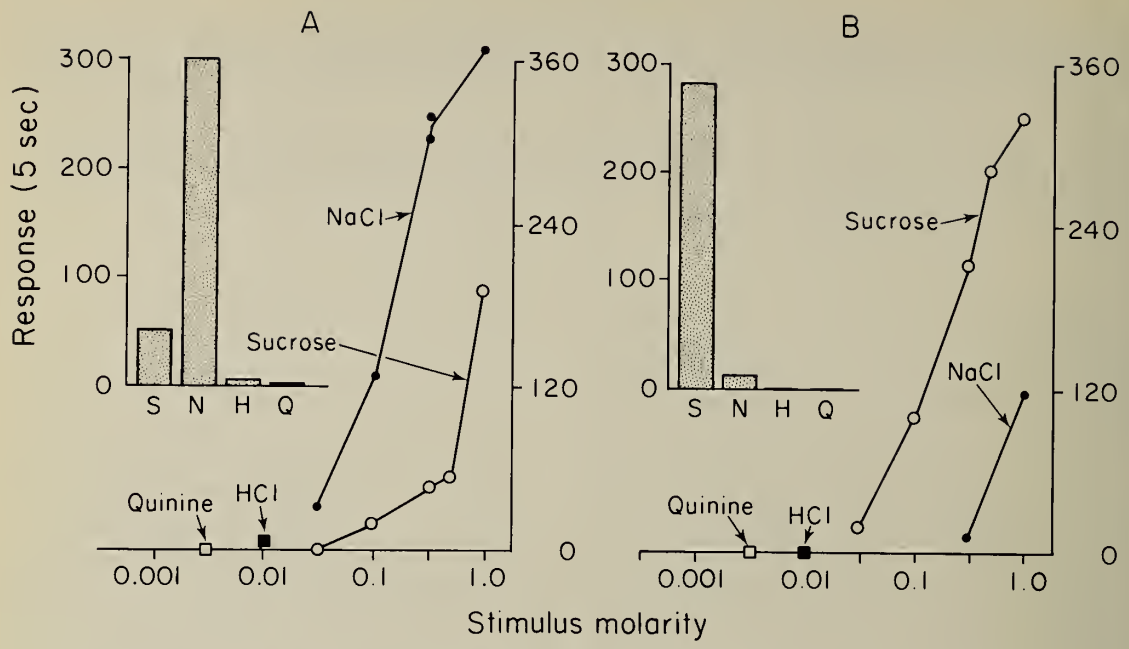


Figure 2–2. The response (number of nerve impulses in 5 sec.) of two (A and B) squirrel monkey chorda tympani nerve fibers as a function of $\log_{10}$ molarity of four tastes: sucrose, NaCl, HCl, and quinine hydrochloride. Insets are response profile histograms for test molarities: 0.5 sucrose (S), 0.3 NaCl (N), .01 HCl (H), and .003 quinine (Q). (From Pfaffmann, Frank, Bartoshuk, and Snell,[7] with permission of Academic Press)

ously to stronger stimuli. However, some compounds are much more effective in eliciting responses in an individual fiber than other compounds throughout the dynamic intensity range. Which compound is more effective, and how much more effective it is, depends on the particular fiber sampled.

Response profiles based on the best of four tastes

Instead of representing response profiles of each nerve fiber in individual sets of bar graphs; comparable profiles can be plotted as line graphs (each line connecting response points of one fiber) and superimposed one over the other and, thus, immediately compared. The profiles of the five hamster chorda tympani fibers which, of the 79 sampled, gave the largest responses to A, sodium chloride; B, hydrochloric acid; C, sucrose; and D, quinine hydrochloride are represented in Figure 2–3.[3] Each line represents the response profile across the test intensities of sucrose (*S*), sodium chloride (*N*), hydrochloric acid (*H*), and quinine (*Q*).

The fibers that, relative to the other fibers, give the largest response to salt respond better to salt than to any of the other tastes. Their second best response is to acid. Similarly, the largest responses to acid come from acid best fibers, and these fibers respond second best to salt. However, four of the five fibers most sensitive to quinine respond better to acid than to

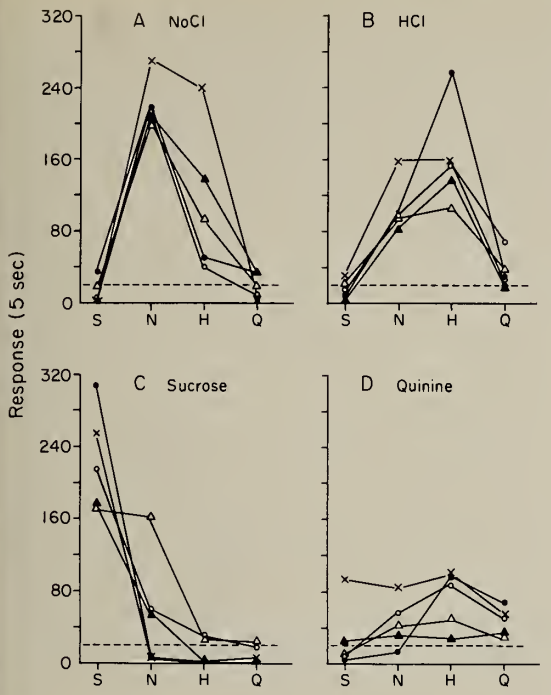


Figure 2–3. Response profiles across four tastes: 0.1 M sucrose (S), .03 M NaCl (N), .003 M HCl (H), and .001 M quinine hydrochloride (Q) of 20 hamster chorda tympani nerve fibers. These profiles are for 5 fibers which responded most strongly to NaCl (A), HCl (B), sucrose (C), or quinine (D) of 79 fibers sampled. The response measure is the number of nerve impulses in the first 5 sec. of response. The dashed line at a response level of 20 represents an arbitrary threshold. (From Frank,[3] with permission of The Rockefeller University Press).

quinine. The five fibers most sensitive to sucrose all give their best response to sucrose, one of these giving a nearly equal response to salt.

Figure 2–4 shows comparable response profiles of squirrel monkey chorda tympani fibers.[4] These are profiles for 20 of the more sensitive of the 45 fibers sampled and are grouped according to which one of the four tastes elicited their largest (or best) response. Fibers in the same category, either salt-best (A), acid-best (B), or sucrose-best (C), have similar response profiles. Profiles for two fibers which do not show one of the three more typical response patterns, along with profiles for three quinine-best fibers, are also plotted (D). Note the relatively specific response to sucrose of four of five fibers in the sucrose-best category and the one fiber of the group which responds equally to sodium chloride, a result nearly identical to that which obtains

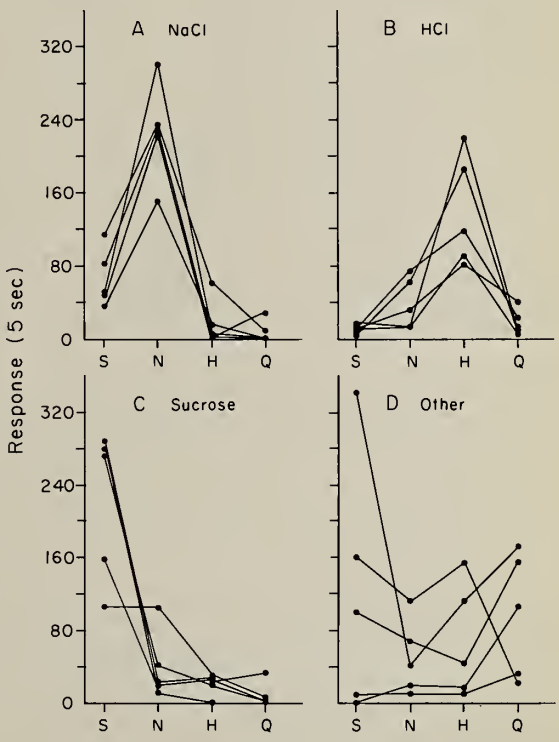


Figure 2–4. Response profiles across four tastes: 0.5 M sucrose (S), 0.3 M NaCl (N), .01 M HCl (H), and .003 M quinine hydrochloride (Q) of 20 squirrel monkey chorda tympani nerve fibers. These profiles are for the more sensitive of 45 fibers sampled which responded best to (A) NaCl, (B) HCl, (C) sucrose, or (D) quinine of the four tastes; or, also (D), showed unusual profiles. The response measure is the number of nerve impulses in the first 5 sec. of response. (From Frank,[4] with permission of D. Reidel Publishing Co.)

for hamster sucrose-best chorda fibers.

If responses of the chorda tympani fibers of each species which respond best to sucrose of the four tastes are averaged, and likewise, responses for fibers which respond best to salt or acid; mean response profiles result. If, however, responses of each fiber are first expressed as a percentage of the fiber's best response, the average percentage of best response profiles in Figure 2–5 result.[4] Too few quinine-best fibers are sampled from either nerve to warrant an average response profile. The mean relative responses of sucrose-best fibers of the chorda tympani nerves of both species across the four tastes are nearly identical. Sucrose-best fibers respond second-best to salt and very little to acid or quinine. However, where salt-best fibers of the squirrel monkey respond second-best to sucrose; hamster salt-best fibers respond second-best to acid. The mean relative response of squirrel monkey salt-best chorda fibers to sucrose is half the size of the response to salt; for hamster fibers in the same category it is only a quarter. Squirrel monkey salt-best fibers, therefore, contribute more to a response to sucrose than do hamster salt-best fibers.

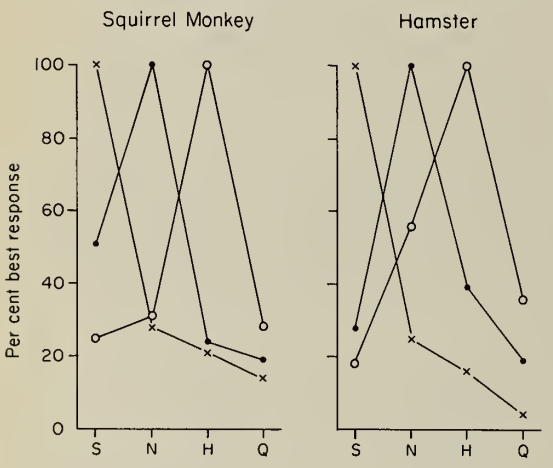


Figure 2–5. Mean relative response profiles of squirrel monkey and hamster chorda tympani nerve fibers across four tastes: 0.5 M sucrose (S), 0.3 M NaCl (N), .01 M HCl (H), and .003 M quinine hydrochloride (Q) for squirrel monkey; and 0.1 M sucrose, .03 M NaCl, .003 M HCl, and .001 M quinine hydrochloride for hamster fibers. The nerve fibers of each species which responded best (greatest number of impulses in 5 sec.) to sucrose (squirrel monkey, n=15; hamster, n=20), NaCl (squirrel monkey, n=11; hamster, n=42), or HCl (squirrel monkey, n=10; hamster, n=17) of the four tastes were grouped together. Responses of each fiber to the four tastes were expressed as percentages of the best response and the mean percentages for each group plotted. (From Frank,[4] with permission of D. Reidel Publishing Co.)

Profiles which include responses to other stimuli

Responses to two additional stimuli were included in hamster chorda tympani profiles and Figure 2–6 shows the results: profiles for the eight most responsive sucrose-best,A, and salt-best,B, fibers which were also tested with 0.3 M fructose (*F*) and .03 M ammonium chloride (*A*).[4] The stimuli are ordered along the abscissa according to their effectiveness in sucrose-best fibers. The heavier line connects mean points. Fibers in the same response category have similar profiles across six stimuli as they did across four. Hamster sucrose-best fibers are rather specific for sweets; fructose eliciting the second-largest mean response and the response to sodium chloride, the third largest, being the only other of considerable size. Hamster salt-best chorda fibers respond second-best to acid and only

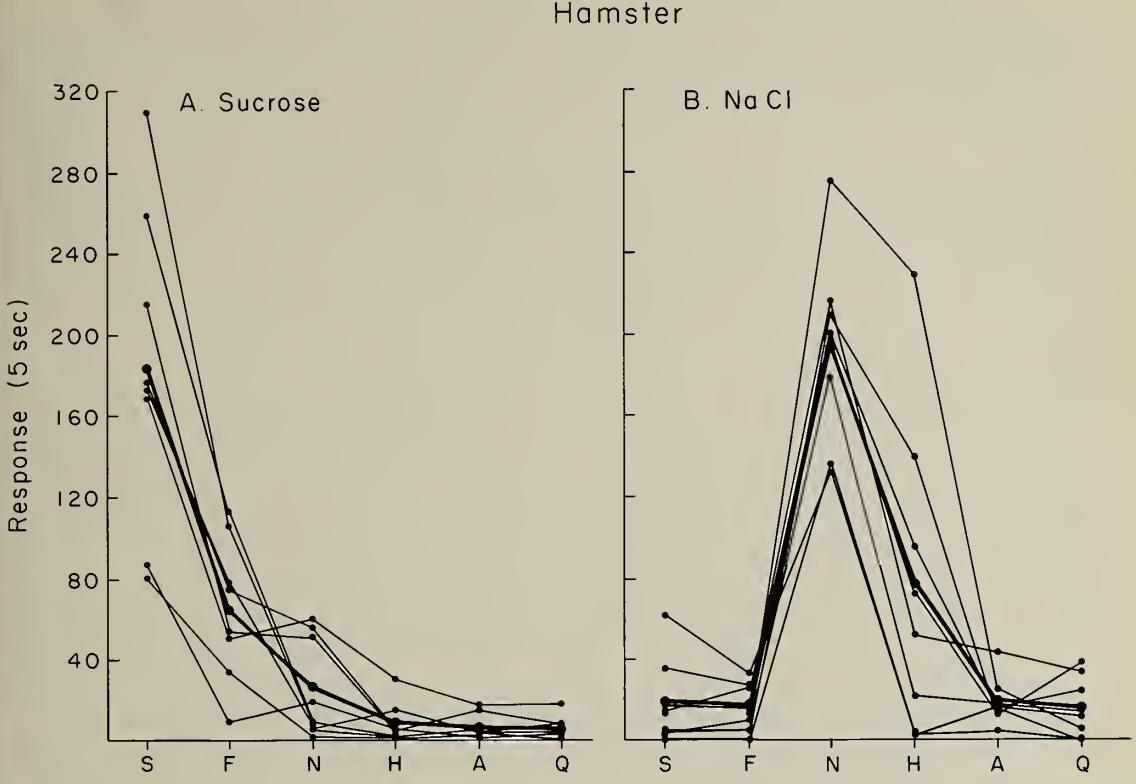


Figure 2–6. Response profiles across six tastes: 0.1 M sucrose (S), 0.3 M fructose (F), .03 M NaCl (N), .003 M HCl (H), .03 M NH_4Cl (A), and .001 M quinine hydrochloride (Q) of 16 hamster chorda tympani nerve fibers, 8 of which responded best to (A) sucrose, and 8 to (B) NaCl of the four tastes: sucrose, NaCl, HCl, or quinine. The fibers most sensitive to sucrose (A) or NaCl (B) are plotted of totals of 11 sucrose-best and 24 salt-best fibers sampled. The response measure is the number of nerve impulses in the first 5 sec. of response. The points connected by heavier lines are means. The mean order of effectiveness of stimuli in the sucrose-best fibers determined their order along the abscissa. (From Frank,[4] with permission of D. Reidel Publishing Co.)

slightly to the other four stimuli.

Comparable plots (including responses to the additional compounds) for squirrel monkey sucrose- and salt-best chorda tympani nerve fibers are shown in Figure 2–7.[4] Again, fibers in the same category have similar profiles. However, squirrel monkey sucrose-best fibers respond better to fructose than do hamster fibers in this category. In fact, both sucrose- and salt-best squirrel monkey chorda fibers respond well to fructose (0.5 M). The two sugars (sucrose and fructose), however, have different relative effectivenesses in the two categories of squirrel monkey nerve fibers. Sucrose is more effective than equimolar fructose in sucrose-best fibers; but fructose is the more effective of the two in salt-best fibers. Also, the salt-best fiber of the squirrel monkey responds very well to ammonium chloride (0.3 M). Ammonium chloride is, in fact, more effective than equimolar sodium chloride for

most squirrel monkey salt-best fibers. Hamster salt-best fibers do not respond well to either fructose or ammonium chloride.

Summary

Examination of response profiles for hamster and squirrel monkey chorda tympani fibers shows that they are broadly tuned across quality and that their sensitivities vary quantitatively from one fiber to the next. Both of these findings are in accord with the classical view of taste fiber sensitivities. However, clusters of sensitivities commonly appear together in

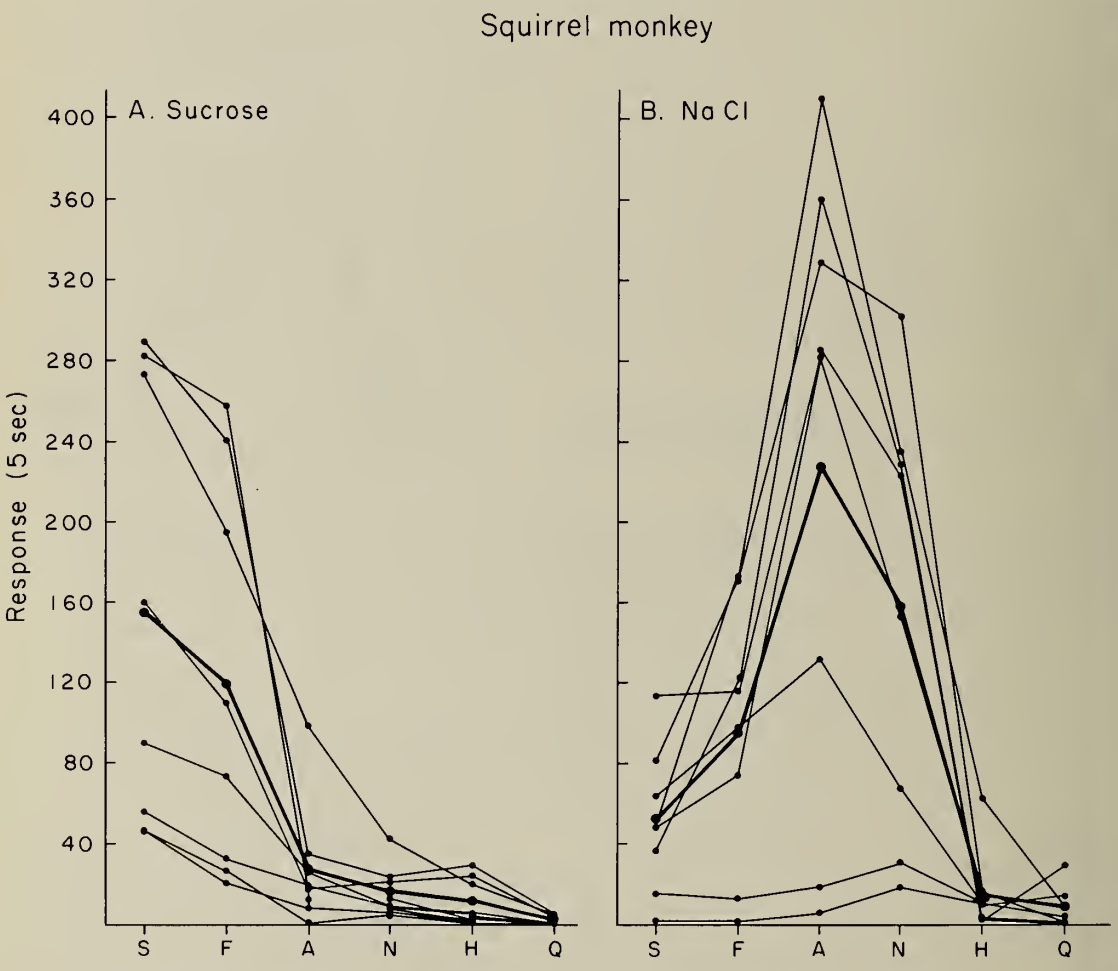


Figure 2–7. Response profiles across six tastes: 0.5 M sucrose (S) and fructose (F), 0.3 M NH_4Cl (A) and NaCl (N), .01 M HCl (H), and .003 M quinine hydrochloride (Q) of 16 squirrel monkey chorda tympani nerve fibers, 8 of which responded best to (A) sucrose and 8 to (B) NaCl of the four tastes: sucrose, NaCl, HCl, or quinine. The fibers whose profiles are plotted are those most sensitive to sucrose of 11 sucrose-best fibers and the 8 salt-best fibers sampled. The response measure is the number of nerve impulses in the first 5 sec. of response. The points connected by heavier lines are means. The mean order of effectiveness of stimuli in the sucrose-best fibers determined their order along the abscissa. (From Frank,[4] with permission of D. Reidel Publishing Co.)

single fibers of each species although sensitivity clusters differ across species. Also, and in particular, a sensitivity to sweet occurs at its greatest and nearly alone in fibers which respond best to sucrose of four tastes (the sucrose-best). Sensitivity to sweet also occurs, but to a lesser extent, together with a strong sensitivity to salts in the salt-best fibers.

THE DISTRIBUTION OF QUALITY SENSITIVITIES ACROSS FIBERS

Comparison with random distribution

The selective manner in which one sensitivity is distributed among single taste fibers can be demonstrated by comparing the observed numbers of particular combinations of sensitivities with those which would be expected if combinations were random. Table 2–I lists each possible response profile, the number of times it occurs in 79 hamster chorda tympani fibers, and the number of times it would occur if combinations were random.[3] In each case, the response profile letter indicates that a response criterion of three times the spontaneous response rate was reached. Combinations of sensitivities would be random if they could be predicted from the proportions of fibers in the sample showing each individual sensitivity. These proportions were: 0.39 for sucrose or *S*, 0.72 for sodium chloride or *N*, 0.48 for hydrochloric acid or *H*, and 0.18 for quinine or *Q*. For example, the predicted value for the profile $-N--$ is (0.61) (0.72) (0.52) (0.82) × 79 = 14.8 fibers. It is the probability of a fiber in the sample not responding to sucrose (1.00 − 0.39 = 0.61), responding to sodium chloride (0.72), and not responding to hydrochloric acid (1.00 − 0.48 = 0.52) or quinine (1.00 − 0.18 = 0.82) multiplied by the number of fibers in the sample (79). Notice the largest deviations from random combination: (1) a sucrose sensitivity occurs alone too often (there are 9 *S* fibers which respond just to sucrose, only 4 were predicted) and (2) sensitivities to the other stimuli occur together too often (there are 10 *NHQ* fibers which respond to sodium chloride, hydrochloric acid, and quinine, but not to sucrose; only 3 were predicted).

TABLE 2–I

DISTRIBUTION OF HAMSTER CHORDA TYMPANI RESPONSE PROFILES TO FOUR TASTES

Response Profile				*Number of Fibers*	
				Observed	*Predicted*
–	–	–	–	8	5.7
S	–	–	–	9	3.7
–	N	–	–	12	14.8
–	–	H	–	2	5.3
–	–	–	Q	0	1.3
S	N	–	–	9	9.5
S	–	H	–	2	3.4
S	–	–	Q	0	0.8
–	N	H	–	14	13.7
–	N	–	Q	2	3.3
–	–	H	Q	1	1.2
S	N	H	–	9	8.7
S	N	–	Q	0	2.1
S	–	H	Q	0	0.7
–	N	H	Q	10	3.0
S	N	H	Q	1	1.9

A response to sucrose, NaCl, HCl, or quinine is indicated by S, N, H, or Q, respectively, and no response by –.

Consideration of the frequencies with which pairs of sensitivities are combined in single hamster chorda tympani fibers yields the similar result seen in Table 2–II.[3] The predicted number of fibers which would show each random sensitivity combination

TABLE 2–II

DISTRIBUTION OF SENSITIVITIES TO TASTE STIMULUS PAIRS AMONG HAMSTER CHORDA TYMPANI FIBERS

Response Combination	*Number of Fibers*	
	Observed	*Predicted*
Sucrose, NaCl	19	22.2
Sucrose, HCl	12	14.8
Sucrose, Quinine	1	5.5
NaCl, HCl	34	27.3
NaCl, Quinine	13	10.2
HCl, Quinine	12	6.8

is calculated by multiplying appropriate whole sample proportions by the number of fibers in the sample. For example, the number of fibers which would show the sucrose-acid random combination is $(0.39)(0.48) \times 79 = 14.8$; but the number of fibers which were observed to have this pair of sensitivities is 12.

A sensitivity to sucrose combines with each of the other sensitivities too few times; and conversely, the other three sensitivities are paired together too frequently for the distribution to be random. Altogether, there are 10 fewer pairings with sucrose and 15 more pairings of tastes other than sucrose than were predicted. This shows the sucrose sensitivity in hamster chorda tympani nerve fibers to be more distinct than sensitivities to the other three tastes. These three are all less distinct and less separate from each other.

Across fiber response correlations

Table 2–III gives rank-difference correlation coefficients between responses of hamster chorda tympani fibers for the six pairs of the four tastes.[3] The correlations between responses to sucrose and any other taste

TABLE 2–III

RANK-DIFFERENCE CORRELATIONS BETWEEN RESPONSES OF HAMSTER CHORDA TYMPANI FIBERS TO FOUR TASTES

	NaCl	*HCl*	*Quinine*
Sucrose	−0.12	−0.26	−0.11
NaCl		+0.51	+0.41
HCl			+0.58

N = 79, $r_s \geq \pm 0.30$, $p \leq 0.01$, t test.

are uniformly negative and not statistically significant. In contrast, all correlations between responses to pairs of the three other tastes are positive and statistically significant. The negative correlations between a sucrose sensitivity and a sensitivity to each of the other stimuli indicate that sizable sucrose responses occur in fibers whose responses to the other stimuli are all small. The significant positive correlations between responses to the other three tastes indicate that these sensitivities are less likely to occur at their greatest alone. They are less separate from each other than from the sucrose sensitivity. In the hamster chorda tympani, then, the sucrose or sweet sensitivity is the most distinct of sensitivities to taste stimuli which represent man's four taste qualities.

Table 2–IV presents the correlations of responses to sucrose and to hydrochloric acid with responses to all other test stimuli.[3] The correlations are ordered from greatest positive to greatest negative for both sucrose and acid. Sucrose responses are significantly correlated across fibers only with responses to fructose and saccharin, i.e., those stimuli which also taste sweet to man. Responses to bitter, salt, and acid stimuli are progressively more negatively correlated with sucrose responses. In contrast, hydro-

TABLE 2–IV

ORDERED RANK-DIFFERENCE CORRELATIONS BETWEEN RESPONSES OF HAMSTER CHORDA TYMPANI FIBERS TO SUCROSE OR HYDROCHLORIC ACID AND OTHER TASTES

Sucrose		*HCl*	
Fructose	+0.77	Citric acid	+0.82
Saccharin	+0.61	Acetic acid	+0.75
Urea	+0.14	KCl	+0.59
$MgSO_4$	−0.03	NH_4Cl	+0.58
Quinine	−0.11	Quinine	+0.58
NaCl	−0.12	$MgSO_4$	+0.57
KCl	−0.14	NaCl	+0.51
$NaNO_3$	−0.19	$NaNO_3$	+0.49
NH_4Cl	−0.21	Urea	+0.47
Acetic acid	−0.25	Saccharin	−0.11
HCl	−0.26	Fructose	−0.16
Citric acid	−0.36	Sucrose	−0.26

N = 79 for sucrose, NaCl, HCl, and quinine; $r_s \geq \pm 0.30$; or N = 46–49 for other stimuli; $r_s \geq \pm 0.37$; $p \leq 0.01$, t test.

chloric acid responses are significantly positively correlated with responses to all non-sweet stimuli. The order of hydrochloric acid response correlations is nearly opposite the order of sucrose correlations; with sweet stimuli yielding the only negative and statistically non-significant correlations, and the organic acids the most highly positive significant correlations with hydrochloric acid. This illustrates that a general sweet sensitivity, not just the sucrose sensitivity, is more distinct than sour or other non-sweet sensitivities in the hamster chorda tympani.

Taste quality profiles

Similarities in distribution of responses to sweet stimuli and their difference from distributions of responses to salty, sour, and bitter stimuli can be depicted with correlation taste profiles.[3] In such plots, the four tastes are taken as taste quality prototypes and correlations with them as measures of the extent a stimulus is of that quality. In Figure 2–8 note the similarity in profile for the three sweet (sucrose, fructose, saccharin) or for other stimuli which are of the same taste quality to man. The greatest differences are between profiles for sweet and non-sweet stimuli. The quality profiles for non-sweet stimuli are all more like one another than they are like any of the three profiles for sweet stimuli.

DISCOVERING DIMENSIONS OF TASTE QUALITY

Unlike other senses, such as vision or audition, taste has no one obvious physical or chemical stimulus dimension which can be varied to effect changes in perceived quality. R.P. Erickson and his colleagues have analyzed taste nerve fiber responses in attempts to discover the effective stimulus variables.[2,8] The Schiffman-Falkenberg multidimensional scaling technique, which has been focussed on of late, positions both taste stimuli and taste fibers in the same Euclidean taste space. Dimensions of the space are determined by principle component factor analysis of responses of all fibers to all stimuli. Stimuli which affect the same fibers to the same relative extent are placed near to, and stimuli which affect different fibers far from, each other in the space. Fibers are placed closest to stimuli to which they respond most and farthest from stimuli to which they respond least.

Figure 2–9 shows the taste space for responses of 40 hamster chorda tympani nerve fibers to 13 stimuli. This

space is developed via computer with a program provided by S. Schiffman. The two dimensions plotted are the only two uncovered which are rele-

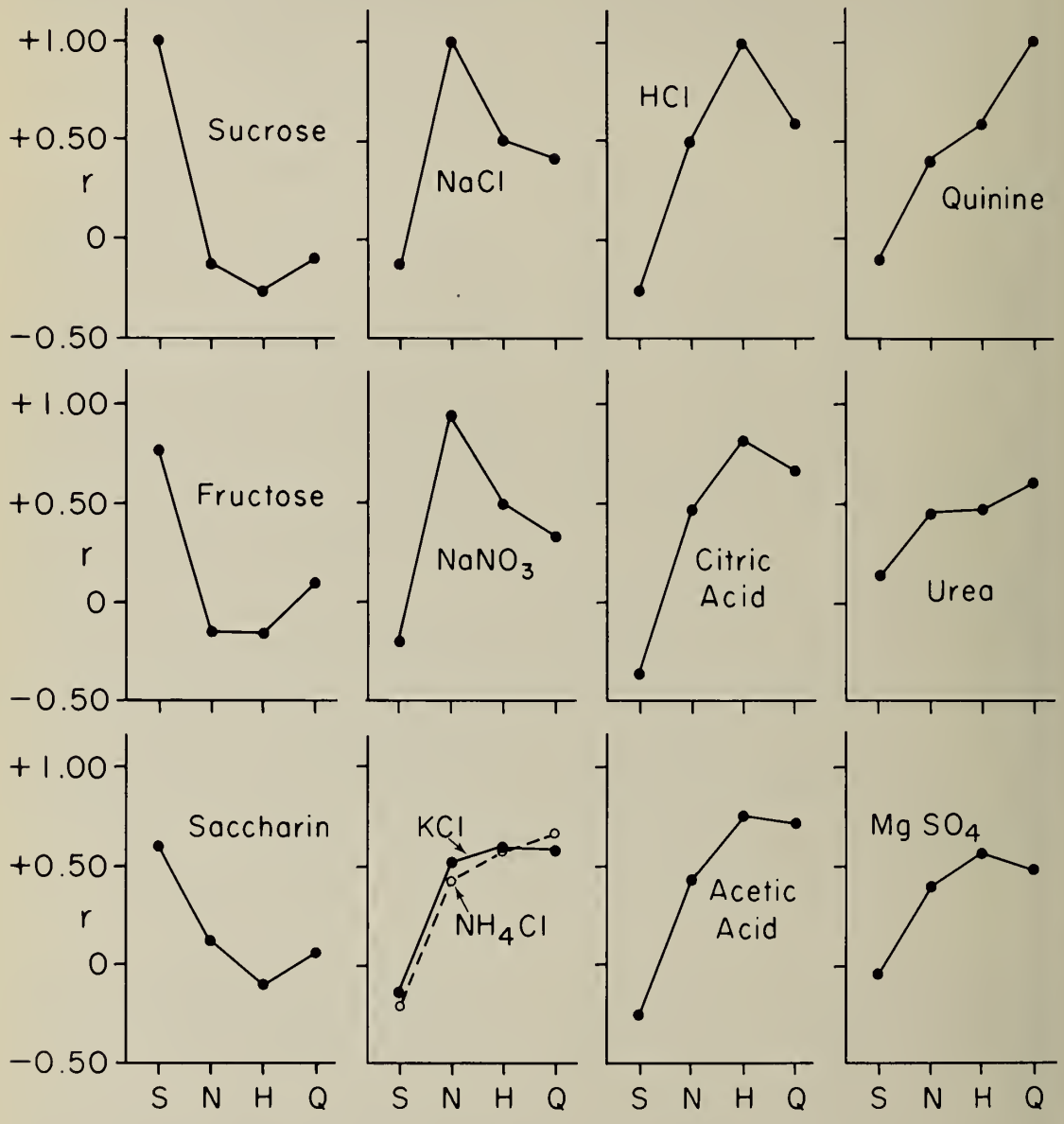


Figure 2–8. Correlation taste profiles across four tastes: 0.1 M sucrose (S), .03 M NaCl (N), .003 M HCl (H), and .001 M quinine hydrochloride (Q) for the responses (number of nerve impulses in 5 sec.) of 46–79 hamster chorda tympani nerve fibers to these tastes and 0.3 M fructose, .001 M Na-saccharin, .03 M $NaNO_3$, .03 M NH_4Cl, 0.1 M KCl, .003 M acetic acid, .001 M citric acid, 1.0 M urea, and .03 M $MgSO_4$. The *r*'s plotted are Spearman rank-difference correlation coefficients. The coefficients for responses to sucrose and sucrose and for each taste and itself were assumed to equal +1.00. (From Frank,[3] with permission of The Rockefeller University Press)

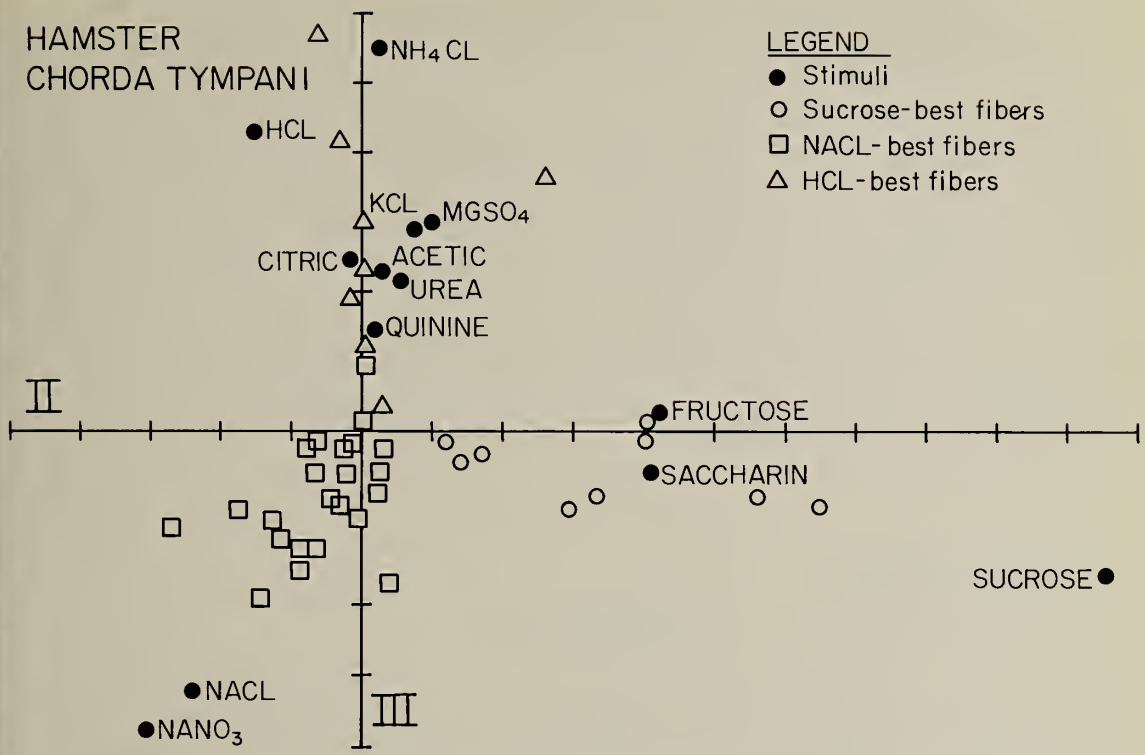


Figure 2–9. The Schiffman-Falkenberg taste space developed from the responses of 40 hamster chorda tympani nerve fibers to 0.1 M sucrose, .03 M NaCl, .003 M HCl, .001 M quinine hydrochloride, 0.3 M fructose, .001 M Na-saccharin, .03 M $NaNO_3$, .03 M NH_4Cl, 0.1 M KC 1, .003 M acetic acid, .001 M citric acid, 1.0 M urea, and .03 M $MgSO_4$. Both the stimuli (represented by solid circles) and the fibers (represented by outline symbols: circles, sucrose-best; squares, NaCl-best; and triangles, acid-best) are plotted in the same Euclidean space. Factor II is plotted along the abscissa, factor III along the ordinate, but factor I, which only accounts for differences in the size of responses across stimuli and fibers, is not plotted. (From O'Connell and Frank, unpublished observations)

vant to taste quality and are statistically significant. Factor II, plotted along the horizontal axis, separates the sweet, especially sucrose, from all non-sweet stimuli. Factor III, plotted vertically, separates sodium salts from other non-sweet stimuli. Fibers are drawn away from each other toward stimuli to which they respond best. They are not found indiscriminately positioned across the space; but are drawn in three directions: (1) toward sucrose, (2) toward sodium salts, or (3) toward acids, non-sodium salts, and quinine. Fibers which respond nearly equally to the various stimuli remain near the origin. The excess of empty space between stimulus and fiber clusters indicates that some patterns of sensitivity occur much more frequently than others in hamster chorda tympani fibers. The great distance sucrose is from the non-sweet stimuli indicates that sucrose, the most effective sweet stimulus, has a highly distinct effect on these fibers.

A comparable plot for about half as many (19) squirrel monkey chorda tympani nerve fiber responses to 10 stimuli, five sugars and five non-sweet stimuli, is seen in Figure 2–10. In the squirrel monkey, as in the hamster, sucrose, the most effective sugar for sucrose-best fibers, is far away from the non-sweet stimuli and nearest to other sugars. Fructose, because of its stronger effect on salt-best fibers, is separated less than sucrose. The squirrel monkey fibers, like those of the hamster, are placed in a few restricted areas and not spread across the whole taste space. Sodium and ammonium chlorides are placed near each other for the squirrel monkey chorda; whereas, for the hamster, the ammonium salt is near hydrochloric acid, not sodium chloride. This is the case because squirrel monkey salt-best fibers respond well to ammonium chloride, whereas in hamster acid-best but not salt-best fibers respond well to ammonium chloride.

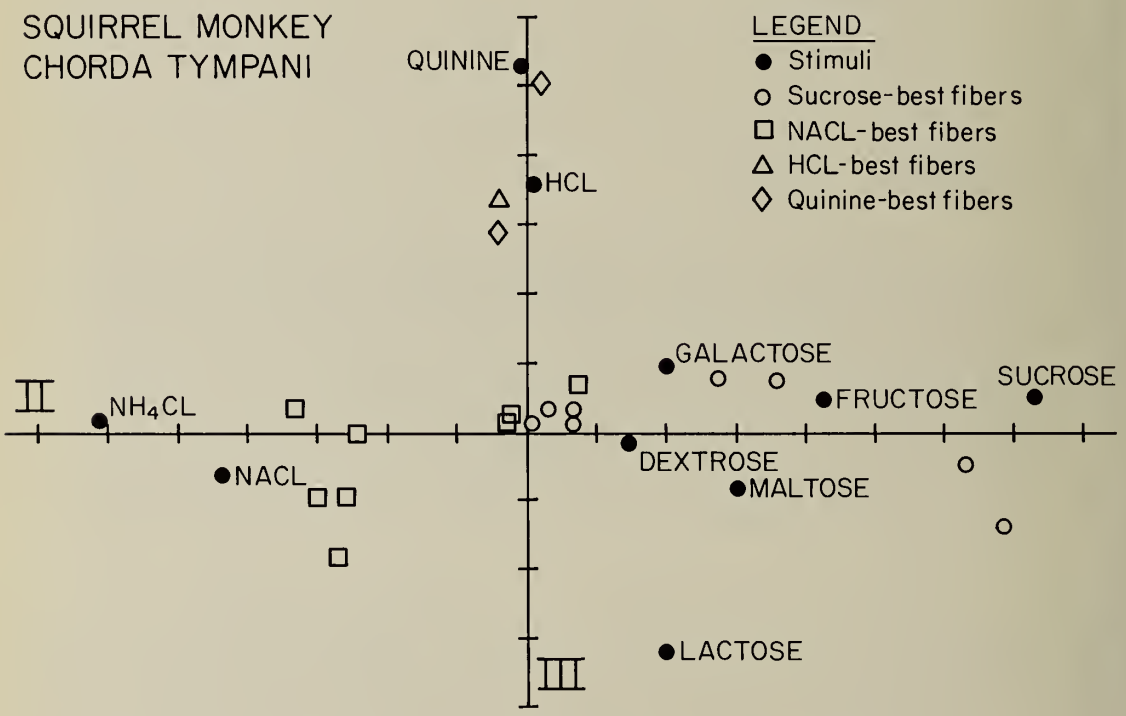


Figure 2–10. The Schiffman-Falkenberg taste space developed from the responses of 19 squirrel monkey chorda tympani nerve fibers to 0.5 M sucrose, fructose, dextrose, galactose, maltose, and lactose; 0.3 M NaCl and NH_4Cl, .01 M HCl, and .003 M quinine hydrochloride. Both the stimuli (represented by solid circles) and the fibers (represented by outline symbols: circles, sucrose-best; squares, NaCl-best; triangles, HCl-best; and diamonds, quinine-best) are plotted in the same Euclidean space. Factor II is plotted along the abscissa, factor III along the ordinate, but factor I, which only accounts for differences in the size of responses across stimuli and fibers, is not plotted. (From O'Connell and Frank, unpublished observations)

CHARACTERISTICS OF THE RESPONSE TO SWEET

Electrophysiological recordings of single peripheral taste nerve fiber responses of the hamster or squirrel monkey chorda tympani show the sweet sensitivity to be unique compared with salt, sour, or bitter sensitivities. That is, stimuli of one of man's quality classes affect individual mammalian nerve fibers in a more selective way than stimuli in any of the other quality classes. The evidence presented supports the following specific conclusions. (1) Single mammalian taste nerve fibers fall into loose species-specific classes, one of which is well-tuned to sweet stimuli. (2) The sweet sensitivity is more distinctly represented in the responses of the chorda tympani fibers than responses to salts, acids, or bitters. And, (3) there are two significant quality dimensions inherent in responses of these chorda tympani fibers and one of these separates sweet from non-sweet stimuli.

REFERENCES

1. Erickson, R.P.: Sensory neural patterns and gustation. In *Olfaction and Taste*. Y. Zotterman (Ed.): New York, Pergamon, 1963, pp. 205–213.
2. Erickson, R.P., Doetsch, G.S., and Marshall, D.A.: The gustatory neural response function. *J. Gen. Physiol.*, 49: 247–263, 1965.
3. Frank, M.: An analysis of hamster afferent taste nerve response functions. *J. Gen. Physiol.*, 61: 588–618, 1973.
4. Frank, M.: The classification of mammalian afferent taste nerve fibers. *Chemical Senses and Flavor,* 1: 53–60, 1974.
5. Ogawa, H., Sato, M., and Yamashita, S.: Multiple sensitivity of chorda tympani fibers of the rat and hamster to gustatory and thermal stimuli. *J. Physiol. (Lond.),* 199: 223–240, 1968.
6. Pfaffmann, C.: Gustatory nerve impulses in rat, cat, and rabbit. *J. Neurophysiol.*, 18: 429–440, 1955.
7. Pfaffmann, C., Frank, M., Bartoshuk, L.M., and Snell, T.C.: Coding gustatory information in the squirrel monkey chorda tympani. In *Progress in Psychobiology and Physiological Psychology.* J. Sprague and A. Epstein (Eds.): New York, Academic, 1976, pp. 1–27.
8. Schiffman, H., and Falkenberg, P.: The organization of stimuli and sensory neurons. *Physiol. Behav.*, 3:197–201, 1968.
9. Tateda, H., and Hidaka, I.: Taste responses to sweet substances in rat. *Memoirs of the Faculty of Science, Kyushu University, Series E,* 4:137–149,1966.

DISCUSSION

MISTRETTA: I would like to know if you looked for differences in temporal patterning of the response for sweet sensitive vs. multi-sensitive fibers. I have observed a bursting pattern in sweet-sensitive fibers in the rat, and also in fibers especially sensitive to acid stimuli.[1]

[1] Mistretta, C.M.: A quantitative analysis of rat chorda tympani fiber discharge patterns. In Schneider, D. (Ed.) *Olfaction and Taste IV,* Wissenschaftlich Verlags. MBH, Stuttgart, 1972, pp. 294–300.

FRANK: Although I've never tried to quantify the effect, I've noticed that bursty responses do occur in hamster and squirrel monkey chorda tympani fibers. They occur most obviously and most frequently in responses to sucrose (or other sweet stimuli) of sucrose best fibers. But they also occur in other fibers. All the responses of a given fiber may be bursty or only a few stimuli may elicit that kind of response. Of the four tastes, quinine and hydrochloric acid also sometimes elicit bursty responses; but responses to sodium chloride are seldom bursty.

As far as thinking of "burstyness" as a temporal code for sweetness, we must take into account that all responses of chorda tympani fibers to sweet stimuli are not obviously bursty.

BRADLEY: It seems to me that you are defining your response in an arbitrary way. In fact the animal may not use such a definition as yours, and use some entirely different criteria when processing the neural data coming from the taste receptors.

FRANK: That's right. The sum of impulses in five seconds may not be exactly what is considered in neural processing; in fact, it probably isn't. It has allowed us to see order in nerve fiber responses to compounds of known properties and also to see that other mammals may have taste quality classes which are rather similar to man's.

Of course we should look at some other measures, such as temporal patterns in responses. It is possible each nerve fiber could carry much more information in its responses than just whether or not it is firing and how strongly it is firing.

PFAFFMANN: That would be a very interesting point to pursue because if there is any differential processing of the information somewhere in the synaptic pathway, you can build a model which will discriminate at the synapse between bursting of impulses versus steady state discharge whichever one gets the presynaptic potentials up high enough to really fire across the gap. It is really worth keeping that in mind because that certainly is one demonstrated firing property that is differential on synaptic transmission.

LE MAGEN: Do you think the brain can discriminate between what you mentioned?

FRANK: Do I think the brain can discriminate between these profiles? Oh, yes.

GRINKER: It is well known that the behavioral response of rats to sucrose is clear and definite. Why then, do you see a differential responsivity to sucrose in your electrophysiological recordings of hamsters, but not in the rat? Do you think this represents a limitation of the factor analysis or loadings, that they fail to separate sucrose responses from the others? Is it possible that another nerve than the chorda tympani in the rat would pull out these differences?

FRANK: Responses of most rat chorda tympani nerve fibers to sucrose are small and very few fibers respond best to sucrose. In contrast, many hamster chorda fibers repond very strongly and best to sucrose. This is why sucrose *is not* an important element in the multidimensional scaling of rat chorda fiber responses to a variety of stimuli; and why it *is* in the scaling of hamster chorda fiber responses to the stimuli. The positions of sucrose in the taste spaces reflects the different sets of data. It remains

near the origin with other relatively ineffective stimuli in the rat space and is pulled far from the origin as other effective stimuli with selective effects in the hamster space.

The fact that rats can make discriminate behavioral responses to sucrose is not strongly reflected in the rat chorda neural taste space. However, the chorda is not the only peripheral nerve mediating taste. Nerve fibers in the glossopharyngeal, which subserve taste buds on the back of the rat's tongue, were not in the sample used to generate the space. These fibers include some that respond well to sucrose and which must give the rat more information on which to base taste discriminations. But, considering only typical samples of chorda tympani fibers, the sucrose stimulus should be less discriminable for rat than for hamster.

PFAFFMANN: Is that because the magnitude of response is not big enough to permit the quantitative treatment or because it really is less discriminate?

FRANK: With regard to these taste spaces, absolute sizes of responses are much less important than relative sizes of responses to one compound as compared to other compounds. The absolute effectivenesses of stimuli and sensitivities of units are "factored out." Factor I, the intensity factor, is not plotted. The "quality factors" which are plotted deal with variance in the data which is due to other than intensity effects. A stimulus has to affect some fibers in a similar relative way and other fibers in a different relative way to be drawn very far away from the origin.

PFAFFMANN: In other words, if units are truly similar to each other, but respond well, they are plotted close to the origin and that really means they are indiscriminate. But if they are low responders and close to the origin, you can't tell what that means.

FRANK: Yes, in a way. When you consider the positions of units in the space, fibers which gave large responses and are close to the origin are found to be those which responded very well to all stimuli and were not discriminating differences in stimuli. Yet, fibers which did not give large responses to any of the stimuli would also be close to the origin. In this case, responses to all stimuli are small and again there is no discrimination of differences in stimuli.

The same holds for stimulus positions in the space. Those which affect all units similarly would be positioned nearer to the origin than other stimuli having greater differential effects. Two kinds of stimuli are positioned near the origin: general stimuli (those which affect all units to the same relative extent), and ineffective stimuli.

WEIFFENBACH: Are those oblique solutions? Did you rotate?

FRANK: These are unrotated orthogonal factors. The method used by the Schiffman-Falkenberg computer program is Hotelling's iterative method for principle components.

Chapter 3

DOES SACCHARIN HAVE A DUAL TASTE FOR THE RAT?

G. Rolfe Morrison and Alan Jessup

Substances which have, to the human observer, the common property of a sweet taste may produce quite different effects on rats. Sucrose, other common sugars and saccharin are all well known to be highly palatable.[3,13,14,18,19] Among the amino acids, glycine and DL-alanine are also palatable.[4] The rat however appears indifferent to dulcin at all concentrations [2] and has been shown to reject sodium sucaryl [11] and lead acetate.[9]

The high degree of acceptability found for saccharin has led to the assumption that the rat, like the human find this substance sweet—sweet being defined as having a taste similar to that of glucose. It is possible however for saccharin to be palatable for the rat without being sweet. For many of the studies that have employed saccharin its exact taste is largely irrelevant, it has been used as a non-nutritive substance which is highly reinforcing. There are however a number of experiments where the results may be subject to different interpretations depending in part on whether or not saccharin tastes similar to sugar.

Although non-nutritive saccharin is not physiologically inert, changes in blood sugar level [16] and free fatty acid levels [12] following saccharin ingestion have been reported and prior saccharin drinking potentiates insulin induced coma.[17] Are these effects due to some direct postingestional effect of saccharin or are they mediated by its taste? The latter interpretation makes the assumption that sweet tasting stimuli may result in a release of insulin either through an innate reflex or through prior conditioning where the taste of sugar has typically been followed by a rise in blood sugar. It has also been found that prior ingestion of saccharin causes a temporary inhibition of glucose intake with the suggestion that the sweet taste of saccharin is the stimulus for a conditioned satiety effect.[5] For these experiments then the question of whether saccharin actually tastes sweet becomes more interesting.

Two results that have been reported suggest the possibility that the taste of saccharin may be unlike that of sugar. Tapper and Halpern [15] found that a conditioned aversion to saccharin resulting from x-ray induced toxicosis generalizes strongly to glycine and DL-alanine but only slightly to glucose, while an aversion to DL-alanine generalizes to saccharin but not to glucose. The difference be-

Acknowledgment: Supported by grant APO 97 from the National Research Council of Canada.

tween the tastes of saccharin and glucose suggested by these results may be either quantitative, that is due to different degrees of sweetness or qualitative, sweet versus non-sweet.

Kakolewski and Valenstein [6] have shown that diabetic rats reject saccharin while continuing to accept glucose. If, as they suggest this is a conditioned aversion due to the association between the taste of saccharin and the malaise produced by diabetes then the failure to generalize to glucose may again be due to either qualitative or quantitative differences. If however as has been suggested [1] this aversion is unconditioned then the results would seem to be more consistent with a qualitative difference unless the saccharin is substantially sweeter than the glucose, with the rats continuing to accept moderately sweet stimuli but rejecting intensely sweet ones.

In order to investigate the question of what saccharin actually tastes like to the rat we had planned to use a procedure similar to that used by Morrison [10] to compare the tastes of various salts. Since this method is very time-consuming unless you have some idea what you are looking for in advance, we decided to do some preliminary investigations using a much simpler technique. This present report is concerned with the results of these preliminary studies.

EXPERIMENT 1

If a rat is exposed to a novel testing solution his intake is suppressed compared to that of a familiar solution of equal palatability. With increasing familiarity one finds increasing intake a phenomenon that has been termed learned safety.[8]

In this first experiment we planned to make use of this phenomenon in the following way. If different groups of rats were first familiarized with different flavours and then tested for saccharin intake we would expect to find that the amount of saccharin ingested would vary directly with the similarity in taste between saccharin and the pre-exposed solution and thus give us a rough guide to this degree of similarity. As the first step, to check on the validity of the method, we planned to show that we could rep licate the Tapper and Halpern [15] finding that saccharin was similar to glycine but different from glucose.

The procedure was as follows.

32 rats were first adapted to a fluid deprivation schedule where they were allowed to drink water for 20 minutes a day in the test situation, followed by an additional 40 minutes in their home cages. Following this period they were divided into four groups of eight rats each.

All four groups continued on the same drinking schedule for the next 5 days.

Group W rats continued to receive water in the test situation, Group S rats received .005 M sodium saccharin, Group G, 1M Glucose and Group Gy, .2M Glycine.

Following this familiarization period a test day was given in which all rats were given .005 M sodium saccharin for 20 minutes.

Results

Based on the Tapper and Halpern [15] results which appeared to show that

saccharin was similar in taste to glycine but different from glucose we expected to find that the group S and Gy rats who had become familiar with the taste of saccharin or glycine would ingest about the same amount of saccharin on the test day and significantly more than the group W and G rats. In fact there were no differences in mean intake between groups S, Gy, and G and intakes in these three groups were significantly higher than those in group W.

Thus in contradistinction to the prediction from the Tapper and Halpern [15] finding it appears that familiarization to glucose is just as effective as that to saccharin or glycine in elevating saccharin intake.

EXPERIMENT 2

Since the above procedure did not result in an outcome consistent with Tapper and Halpern [15] it was decided that before proceeding we should attempt to replicate their finding of little or no generalization of a conditioned aversion between saccharin and glucose.

An additional 32 animals were first adapted to the water drinking schedule as before and then subdivided into four groups of eight each. Two groups GN and GP were then given .5M glucose for 20 minutes on one day, the remaining two, SN and SP .005M saccharin. Following this drinking period groups GP and SP were poisoned with an IP injection of lithium chloride. On the next day all animals were allowed to drink water once more and then this was followed by a test day on which all animals were given the saccharin solution to drink.

Results

Again based on the Tapper and Halpern [15] results we had expected the animals who had been poisoned following glucose drinking to behave like the unpoisoned controls in the saccharin test. Once again however this prediction was not verified.

Saccharin intake on the test day was virtually equal for groups GP and SP (means: 3.6 and 3.8 ml.) with both groups showing substantial suppression compared with the control groups GN and SN (means: 10.4 and 11.8 ml.). Water intakes for all four groups on the post poisoning day were at their prepoisoning levels indicating that the decreased saccharin intake shown be the poisoned rats was not due to the unconditioned effects of the poisoning.

These results were consistent with those of the first experiment in suggesting that glucose and saccharin are in fact similar in taste.

Thus in two experiments we had failed to confirm Tapper and Halpern's [15] findings on the dissimilarity of glucose and saccharin. They however poisoned their animals following saccharin drinking and tested them on glucose while we had done the reverse. Possibly this difference is critical.

EXPERIMENT 3

2 more groups of eight rats were run through the same procedure as in

the last experiment. One group (GP) was poisoned following glucose drinking the other (SP) following saccharin drinking. On the test day both groups were given glucose.

Results

On the poisoning day group GP drank an average of 7.1 ml. of glucose and group SP an average of 4.4 ml. of saccharin (These rats were smaller than those of experiment 2 and did less drinking, their average water intake was 6 ml. compared to 9 ml. in experiment 2). On the test day group GP intake declined to 2.6 ml. showing the usual conditioned aversion. Group SP however averaged 6.4 ml. on the test day, a value not significantly different from the prepoisoning glucose intake of group GP.

In other words, the conditioned aversion to saccharin showed little if any generalization to glucose and confirms the Tapper and Halpern [15] results.

DISCUSSION

At this point then we are left with two experiments that suggest that glucose and saccharin are similar in taste for the rat (experiments 1 and 2 above) and two others that suggest they taste quite different (experiment 3 and Tapper and Halpern [15]). Can these differences be reconciled?

One possibility that seems to be worthy of further investigation is that saccharin has a dual taste for the rat, sweet and something else—we would like to suggest bitter but we have absolutely no evidence for this yet. The "something else" however we suggest is aversive.

In the poisoning situation, when the rat is poisoned following saccharin the aversion conditions to the aversive component of the taste, not to the sweet components. This is consistent with the role of stimulus salience in this type of conditioning.[7] Since the glucose used in the test does not have this aversive component there is no generalization of the conditioned aversion. When the animal is poisoned following glucose the conditioning is to sweet since that is the only taste present and this aversion generalizes to the sweet component of the saccharin.

This explanation would account for the asymmetrical results of the above experiments, and might also suggest why diabetic rats shown an aversion to saccharin but not to glucose.

Assume that diabetes causes a change in the sweet sensitive receptors, possibly because these are kept in a state of partial adaptation due to high blood glucose levels. This would mean that glucose would be a less effective stimulus, it is still sweet but less so than for the normal rat, and the sweet preference is still there. When the animal is given saccharin however the receptors sensitive to the sweet component are adapted but those to the other, aversive, one are not. This would result in a taste *quality* change for saccharin with the aversive component becoming more significant in the control of behaviour.

One test of this idea that we are in the process of trying is the following.

If a conditioned aversion to glucose is well established and then the rats are made diabetic by alloxan injection, then if saccharin is no longer primar-

ily sweet there should be no generalization of this conditioned aversion. This would predict that preconditioned diabetic rats and unconditioned ones should show the same reaction to saccharin. They will have an *unconditioned* aversion [1,6] but no conditioned one. If the aversion to glucose is strongly conditioned then these diabetic animals should show greater saccharin intake than glucose conditioned normal ones, the unconditioned aversion in the former being weaker than the generalized conditioned aversion in the latter.

If this prediction is borne out then eventually we may return to our original intention of finding out what saccharin does taste like to the rat.

REFERENCES

1. Brookshire, K.H., Stewart, C.N., and Bhagavan, H.N.: Saccharin aversion in alloan-diabetic rats. *Journal of Comparative and Physiological Psychology*, 79: 385–393, 1972.
2. Fisher, G.L., Pfaffmann, C., and Brown, E.: Dulcin and saccharin taste in squirrel monkeys, rats and men. *Science*, 150:506–507, 1965.
3. Guttman, N.: Equal reinforcement values for sucrose and glucose solutions compared with equal sweetness values. *Journal of Comparative and Physiological Psychology*, 47:358–361, 1954.
4. Halpern, B.P., Bernard, R.A. and Kare, M.R.: Amino acids as gustatory stimuli in the rat. *Journal of General Physiology*, 45:681–701, 1962.
5. Hsaio, S. and Tuntland, P.: Short term satiety signals generated by saccharin and glucose solutions. *Physiology and Behavior*, 7:287–289, 1971.
6. Kakolewski, J.W. and Valenstein, E.S.: Glucose and saccharin preference in alloxan diabetic rats. *Journal of Comparative and Physiological Psychology*, 68:31–37, 1969.
7. Kalat, J.W. and Rozin, P.: "Salience" a factor which can override temporal contiguity in taste-aversion learning. *Journal of Comparative and Physiological Psychology*, 71:192–197, 1970.
8. Kalat, J.W. and Rozin, P.: "Learned safety" as a mechanism in rats' long-delay taste-aversion learning. *Journal of Comparative and Physiological Psychology*, 83:198–207, 1972.
9. Mason, D.J. and Safford, H.R.: Palatability of sugar of lead. *Journal of Comparative and Physiological Psychology*, 59:94–97, 1965.
10. Morrison, G.R.: Behavioural response patterns to salt stimuli in the rat. *Canadian Journal of Psychology*, 21: 141–152, 1967.
11. Murray, E.J., Wells, H., Kohn, M. and Miller, N.E.: Sodium sucaryl: a substance which tastes sweet to humans but is avoided by rats. *Journal of Comparative and Physiological Psychology*, 46:134–137, 1957.
12. Remley, N.R. and Harrell, E.H.: Biological effects of saccharin ingestion in rats. *Psychonomic Science*, 17:295–296, 1969.
13. Richter, C.P. and Campbell, K.H.: Taste thresholds and taste preferences of rats for five common sugars. *Journal of Nutrition*, 20:31–46, 1940.
14. Sheffield, F.D. and Roby, T.B.: Reward value of a non-nutritive sweet taste. *Journal of Comparative and Physiological Psychology*, 43:471–481, 1950.
15. Tapper, D.N. and Halpern, B.P.: Taste stimuli—A behavioral classification. *Science*, 161:708–710, 1968.
16. Thompson, M.M. and Mayer, J.: Hypoglycemic effects of saccharin in experimental animals. *American Journal of Clinical Nutrition*, 7:80–85, 1959.
17. Valenstein, E.S. and Weber, M.L.: Potentiation of insulin coma by saccharin. *Journal of Comparative and Physiological Psychology*, 60:443–446, 1965.

18. Young, P.T. and Greene, J.T.: Relative acceptability of saccharin solutions as revealed by different methods. *Journal of Comparative and Physiological Psychology*, 46:295–298, 1953.

19. Young, P.T. and Shuford, E.H. Jr.: Quantitative control of motivation through sucrose solutions of different concentrations. *Journal of Comparative and Physiological Psychology*, 48:114–118, 1955.

DISCUSSION

COLLIER: I would like to suggest another paradigm. If you make independent sweetness matches between saccharin and sucrose, and offer rats a choice between saccharin and sucrose, the rats will almost inevitably choose sucrose down to a very low concentration (2%) over every concentration of saccharin. There is some difference other than sweetness.

I would like to suggest that the same is true for glucose and sucrose. I wonder how your system would work with that? If you offer a rat sucrose and glucose independently and match them on sweetness, rats will typically prefer sucrose over glucose. I don't know what the "bitter" taste of glucose is, but I think Carl mentioned this phenomenon this morning.

MORRISON: The problem is, of course, what do you mean by sweetness for the rat. In this experiment I am arbitrarily defining sweet as the taste of glucose. I would rather define it as the taste of sucrose but to be comparable with Tapper and Halpern[15] we used glucose. By definition then for the purposes of this study glucose is sweet.

JACOBS: I'm not sure that anyone in human psychophysics would be willing to state that a sweetness judgment to sucrose is the same as a sweetness judgment to glucose. There are also problems associated with taste mixtures including the issue of whether responses are to the parts of a mixture or to their sum. You may want to relate your work to that of Moskowitz and Bartoshuk or do some human experimentation to validate your ideas.

In your control experiment, the simple one, the glucose-trained animals took saccharine on the test day. Would saccharine-trained animals take glucose? In that case, the poison experiment is not really necessary.

MORRISON: There is a practical problem involved here in that, with the glucose concentration we used and the time periods we measured, you don't see a reliable rise in intake from day one to day two. The animals start off taking large amounts of glucose and stay fairly level throughout.

JACOBS: Is your assumption that the post-ingestional effects of glucose account for the changes in threshold or discrimination? I would be pleased if your technique could show this. This would be extremely important for investigators in the field of specific hungers as this is the type of effect of bodily state on sensitivity that they have searched for for decades.

MORRISON: I would suggest that as one possibility that could be considered. At this point we have no data that points to the type of change responsible for the effect. It may be a central change, a receptor change or simply adaptation. I suggested this last one because it would seem to be the simplest and easiest to test.

MISTRETTA: I am reminded of the highly palatable mixture of glucose

and saccharin [1] which rats drink in such large volumes. Perhaps this other saccharin component which you hypothesize is a bitter component, making the solution more palatable. Maybe we really prefer bitter-sweet to pure sweet.

MORRISON: Let me give you another even wilder speculation that did come out in some of the intervening data that would help explain some of the results in the middle period. That is that the bitter—or whatever it is, the unpleasant component—might well be an after-taste, as it is in the human, and if it is, this is going to mean the exact testing situation is going to become very critical.

NOWLIS: I was just puzzling over the problem of matching concentrations across molecular species. In a study [2] using three concentrations of a single taste stimulus, NaCl, I found the following: First, the stronger the salt concentration that was ingested when the poisoning occurred (training concentration), the stronger the aversion to subsequent presentations of salt. Second, the stronger the salt concentration used as a test for aversion (test concentration), the stronger the aversion manifested. Third, there was a multiplicative relationship between the effect on degree of aversion of training concentration and testing concentration. Fourth, above and beyond this multiplicative relationship, there is evidence for a stimulus generalization decrement along the concentration dimension, so that, for example, rats poisoned in conjunction with a weak concentration of salt actually show more acquired aversion to the medium concentration than to the strong concentration. Thus before deciding on the basis of generalization decrement that one sweet substance is not similar to another, one must develop some strategy for addressing the problem of comparability of concentrations. This is not always easy.

MORRISON: All I can say at the moment is that in our studies with the .005 M saccharin we get no generalization, no aversion to 1 M glucose, .5 M glucose, or .1 M glucose. Tapper and Halpern [15] also found no generalization.

On the other hand, going the opposite way, we get complete generalization to that concentration of saccharin. It is a problem. I want to start cross matching concentrations.

[1] Valenstein, E.S., Cox, V.C., and Kakolewski, J.: Polydipsia elicited by the synergistic action of a saccharin and glucose solution. *Science*, 157:552–554, 1967.

[2] Nowlis, G.H.: Conditioned stimulus intensity and acquired alimentary aversions in the rat. *J. comp. physiol. Psychol.*, 86:1173–1184, 1974.

PART II
RESPONSES TO SWEET IN THE FETUS AND SUCKLING YOUNG OF MAMMALS OTHER THAN MAN

Chapter 4

TASTE IN UTERO: THEORETICAL CONSIDERATIONS

Charlotte M. Mistretta and Robert M. Bradley

It is reasonable to expect that genetic and environmental influences interact in the development of taste preference. The challenge for the investigator is to understand the nature and extent of the interaction. The widespread phylogenetic appearance of the 'sweet tooth', as documented by Dr. Pfaffmann, suggests a genetic component in the preference for sweet. Studies on rats and mice suggest that preference for sweet compounds varies with genotype.[33,38] Further, Desor and Maller [19] have shown that from birth human babies prefer sugar solutions to water. In the human, then, it would seem that a preference for sweet is manifest before experiential factors can intervene. However, Dr. Jerome has presented data on the practice, current in a variety of cultures, of exposing infants to sugar from the moment of birth. Mothers throughout the world feed their newborns sweetened forms of liquid diets. The neonate's preference for sweet would be reinforced throughout infancy and afterwards in the many cultures where candy and other sweet foods are given to children (see also Muto). Does the continuous, prolonged exposure of the human to sweetened diets imprint the organism to a food which it prefers from birth anyway? Daily exposure of newborn rats and guinea pigs to some chemicals results in the establishment of a transient preference for the chemical;[26,39] perhaps longer exposures would establish lasting preferences. Galef [22] reports that food habits in rats are transmitted from parent to young. Humans, also, seem to transmit food habits to their infants.

Although these studies identify some of the factors which influence the development of preference behavior, they are not designed to examine the sensory system which underlies the behavior. To understand the contributions of genetic and environmental factors knowledge of the biological substrate on which these factors operate must be complete. It is important to know: when taste buds first appear; when taste receptors are first functional; whether the neurophysiological response characteristics of the taste system change as development proceeds; at what point in neurophysiological development preferences and aversions are first expressed.

Acknowledgment: The authors are grateful for financial support from the Medical Research Council, England (1970–72) and NIH Grant HD07483 (1973 to present).

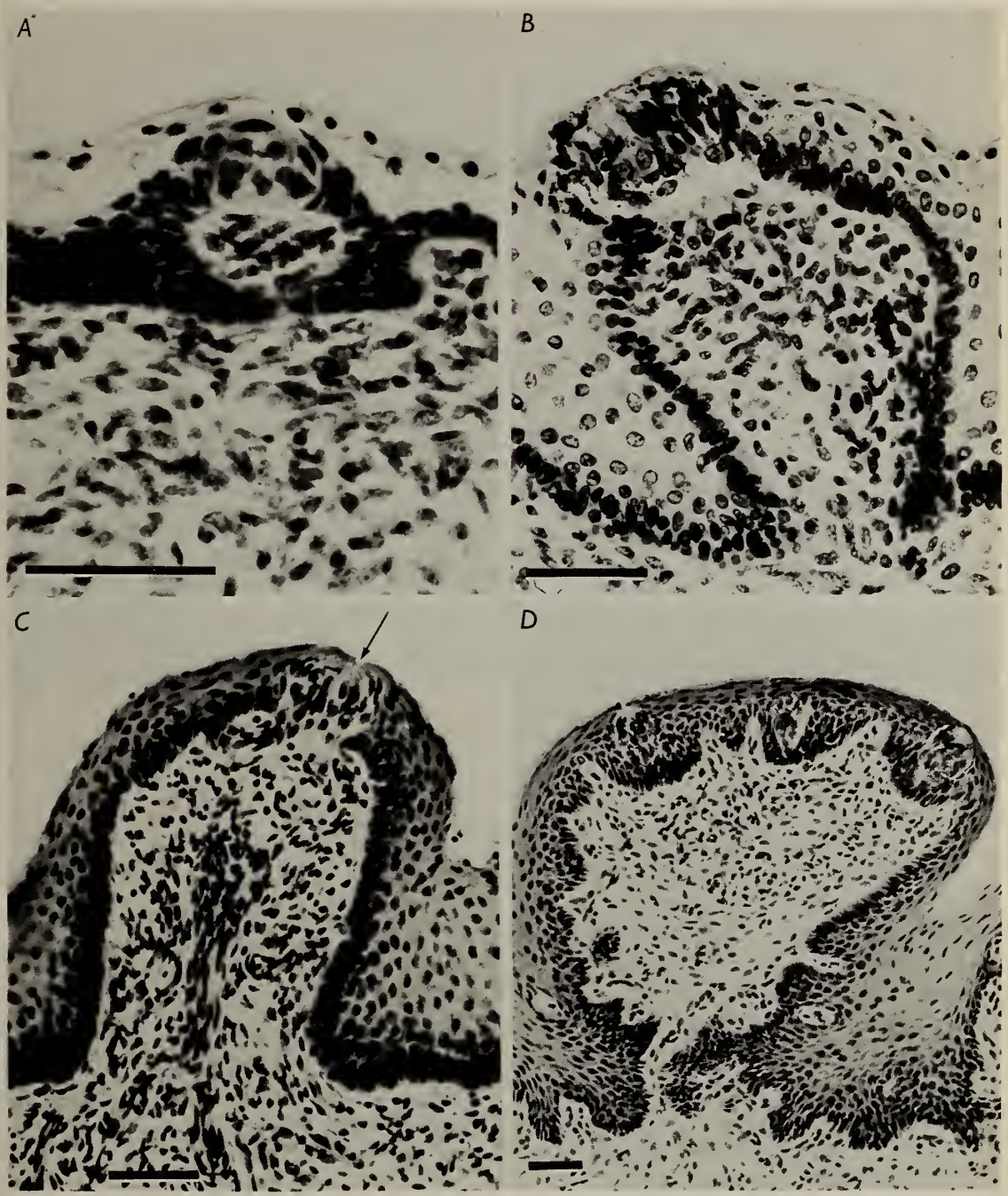
It is known that in humans [6,10] and sheep [7] taste buds on the tongue appear and mature structurally *in utero*. In both of these species taste buds are first seen at about 7–8 weeks of gestation and are morphologically mature by about 14 weeks. Photomicrographs of taste buds from fetal and newborn sheep are presented in Figure 4–1. At 50 days of gestation the presumptive taste bud is composed of disoriented cells, and is still covered with superficial cells of the dorsal tongue epithelium (Fig. 4–1A). There is no direct communication between taste bud cells and the oral cavity. By 84 days the taste bud cells are oriented perpendicularly to the basement membrane and the apical ends of the cells are now exposed to the oral cavity (Fig. 4–1B). By about 100 days of gestation the first distinct signs of a taste pore are found (Fig. 4–1C). Between 50 days and 100 days of gestation only one taste bud is observed per fungiform papilla. Between 100 days and term two buds are frequently found per papilla; in the newborn lamb a single fungiform papilla contains three to four taste buds (Fig. 4–1D). Nerve fibers are found in the connective tissue core of fungiform papillae in 50 day fetal tongues (Fig. 4–2A); by 100 days, individual fibers stain more distinctly and branch more extensively (Fig. 4–2B). The photomicrograph of a fungiform papilla and taste bud from a 14 week human fetus in Figure 4–3 provides a comparison to the fetal lamb papilla in Figure 4–1C.

While the taste buds of the human and sheep are structurally mature before birth, those of the rat reach maturity about 12 days postnatally.[21,31] Since sensory systems are more susceptible to modification by environmental factors during development than when structurally mature [20] species may be differentially effected by variations in the postnatal environment. The taste system of the rat would presumably be more influenced by the postnatal environment than that of the human or sheep. In humans and sheep the sense of taste may be more easily modified by prenatal or intrauterine environmental conditions. But what is known of the functional characteristics of taste receptors before birth? Are taste buds responsive to stimuli *in utero*?

ELECTROPHYSIOLOGY OF THE FETAL TASTE SYSTEM

To determine whether fetal taste buds are functional *in utero* we recorded responses from the chorda tympani nerve while stimulating the tongue of fetal sheep with chemicals.[7] Single and multi-fiber responses were recorded in fetuses aged 96—147 days (term) of gestation. The pregnant ewe was anesthetized with an i.v. dose of sodium pentobarbital, which also effectively anesthetized the fetus. The fetus was delivered onto a warm table at the ewe's side and covered with gauze and cotton. The umbilical

Figure 4–1. Photomicrographs of longitudinal sections through fungiform papillae and taste buds on the anterior third of the sheep tongue at various ages. The scale on each photograph represents 50μ. A. Fungiform papilla and presumptive taste bud in a 50-day fetus. The disoriented cells of the early taste bud are covered by superficial cells of the tongue epithelium. H and E. B. Fungiform papilla and taste bud in an 84-day fetus. The apical ends



of taste bud cells are exposed to the oral cavity. The lightly stained cell in the center of this taste bud should not be confused with a taste pore. H and E. C. Fungiform papilla and taste bud in a 100-day fetus. The papilla and taste bud are covered by superficial cells with flattened nuclei except for a small area over the center of the taste bud (arrow). This is the first distinct sign of a taste pore. H and E. D. Fungiform papilla and three taste buds in a 1-day old lamb. A rather wide taste pit is seen in two of the taste buds. H and E. (From Bradley and Mistretta.[7])

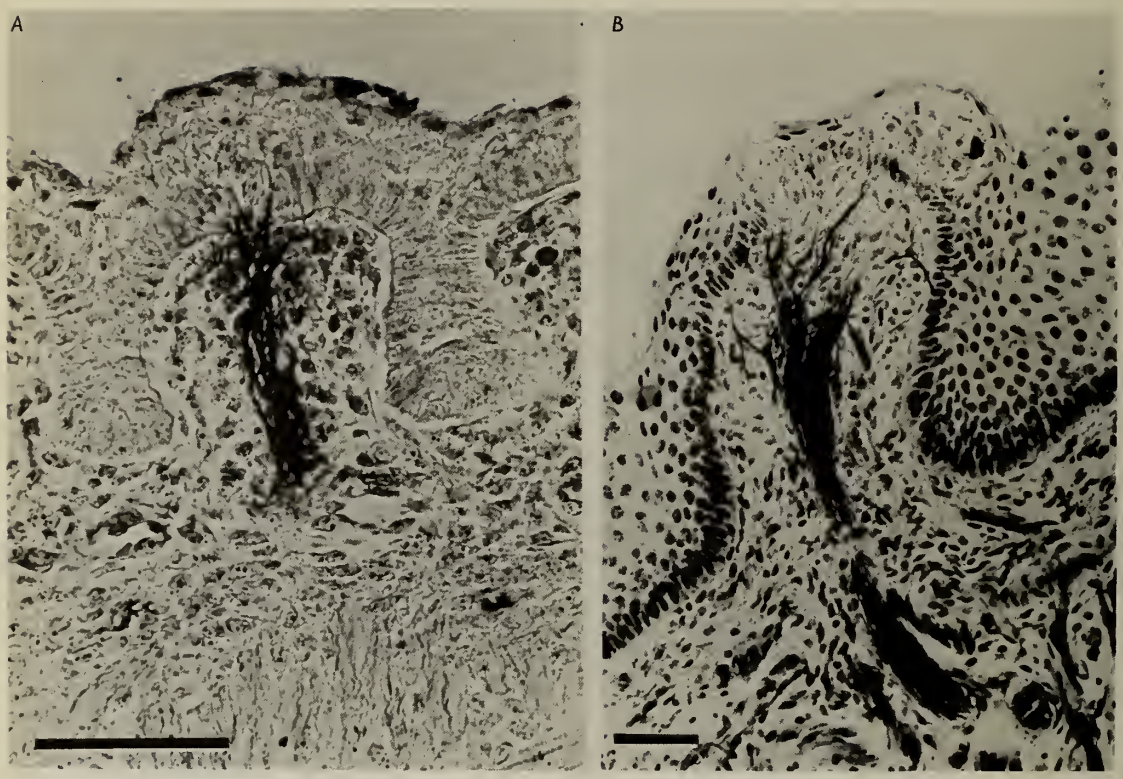


Figure 4–2. A. Fungiform papilla in a 50-day fetus. Nerve fibers penetrate the basement membrane and enter the epithelium in the region where taste buds form. Sevier-Munger. Scale = 50μ. B. Fungiform papilla and two taste buds in a 100-day fetus. The nerve fibers branch extensively and course to cells in both taste buds and to non-gustatory epithelial cells. Sevier-Munger. Scale = 50μ. (From Bradley and Mistretta.[7])

circulation remained intact and fetal and maternal blood gas values and pH were monitored periodically. The fetal head was secured in an atraumatic holder and the chorda tympani was dissected via a lateral approach. Conventional electrophysiological methods were used to record from single fibers, small bundles or the whole nerve. Multi-unit activity was passed through a full-wave rectifier and smoothing circuit with a time constant of 0.5 sec.[5]

Stimuli included 0.05–1.0 M NH_4Cl, 0.5 M KCl, LiCl; and NaCl, 0.5–2.0 M glycerol, fructose, sucrose and glucose, 0.5 M Na saccharin, 0.5 M glycine, 0.5–1.0 M urea, 0.02 M quinine HCl, and 0.01 N HCl. All chemicals were dissolved in distilled water and kept at room temperature during experiments. Stimuli were applied to the tongue using 20 ml plastic syringes. After 30–60 sec the tongue was rinsed with distilled water for at least 30 sec. The stability of each preparation was monitored by periodic stimulation with a standard stimulus, 0.5 M NH_4Cl.

For comparative purposes, records

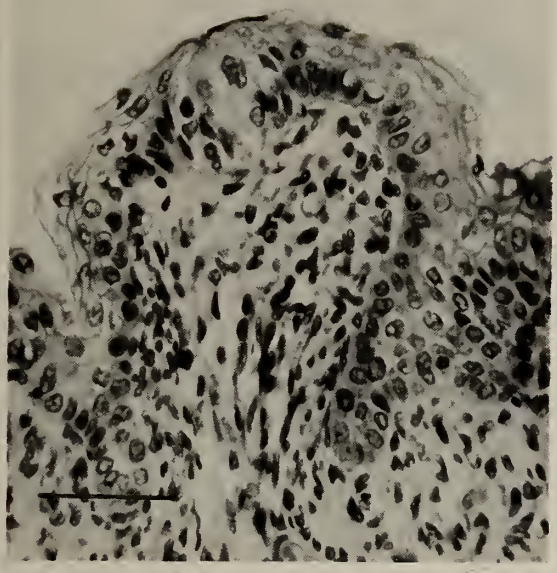


Figure 4–3. Fungiform papilla from the tongue of a 115 mm (~98 day) human fetus. Note taste pore in center of bud. H and E. Scale = 50μ. (From Bradley.[6])

of multi-fiber activity were made from the chorda tympani in newborn lambs and adult ewes.

Over the last third of gestation responses were recorded from the chorda tympani nerve during stimulation of the tongue with a variety of chemicals (Figs. 4–4 and 4–5). All single and multi-fiber preparations responded with a relatively high frequency discharge on application of 0.5 M NH_4Cl to the tongue. Sugars usually did not elicit a response, although glycerol did. In all preparations studied salts and acids elicited a higher frequency neural response than chemicals which taste sweet or bitter to man. Similar responses were obtained from newborn lambs and adults. When a series of 0.5 M monochloride salts (NaCl, KCl, NH_4Cl, LiCl) were applied to tongues of fetuses, the order of effective stimulation was NH_4Cl > LiCl > NaCl = KCl (Fig. 4–6). The same order was found in some, but not all, lamb and adult preparations. Responses to a range of NH_4Cl concentrations (0.1–0.75 M) were recorded in fetuses, lambs, and adults; at all ages, increasing concentrations elicited neural responses of increasing magnitude.

According to the criteria which were used, fetal taste receptors over the last third of gestation responded similarly to those of the newborn and adult. This is perhaps not surprising since the fetal taste buds appear to be morphologically mature by about 100 days. It is important to extend the age-range studied backwards and record from the peripheral taste system in 50–100 day fetuses, during the period when taste buds are maturing. However, the chorda tympani is very fragile in these young fetuses and difficult to dissect without causing damage. Therefore, we have begun to record taste responses in the fetal medulla to chemical stimulation of the tongue. The fetal brain is exposed and a portion of the cerebellum aspirated to reveal the floor of the fourth ventricle. Using coordinates determined from histological sections of fetal brains at different ages, a tungsten microelectrode is positioned in the tractus solitarius. When neural responses to lingual stimulation are recorded, the brain is lesioned at that point to allow subsequent histological confirmation of the electrode position. Chemicals are applied to the tongue as described for the chorda experiments.

Thus far preliminary results have

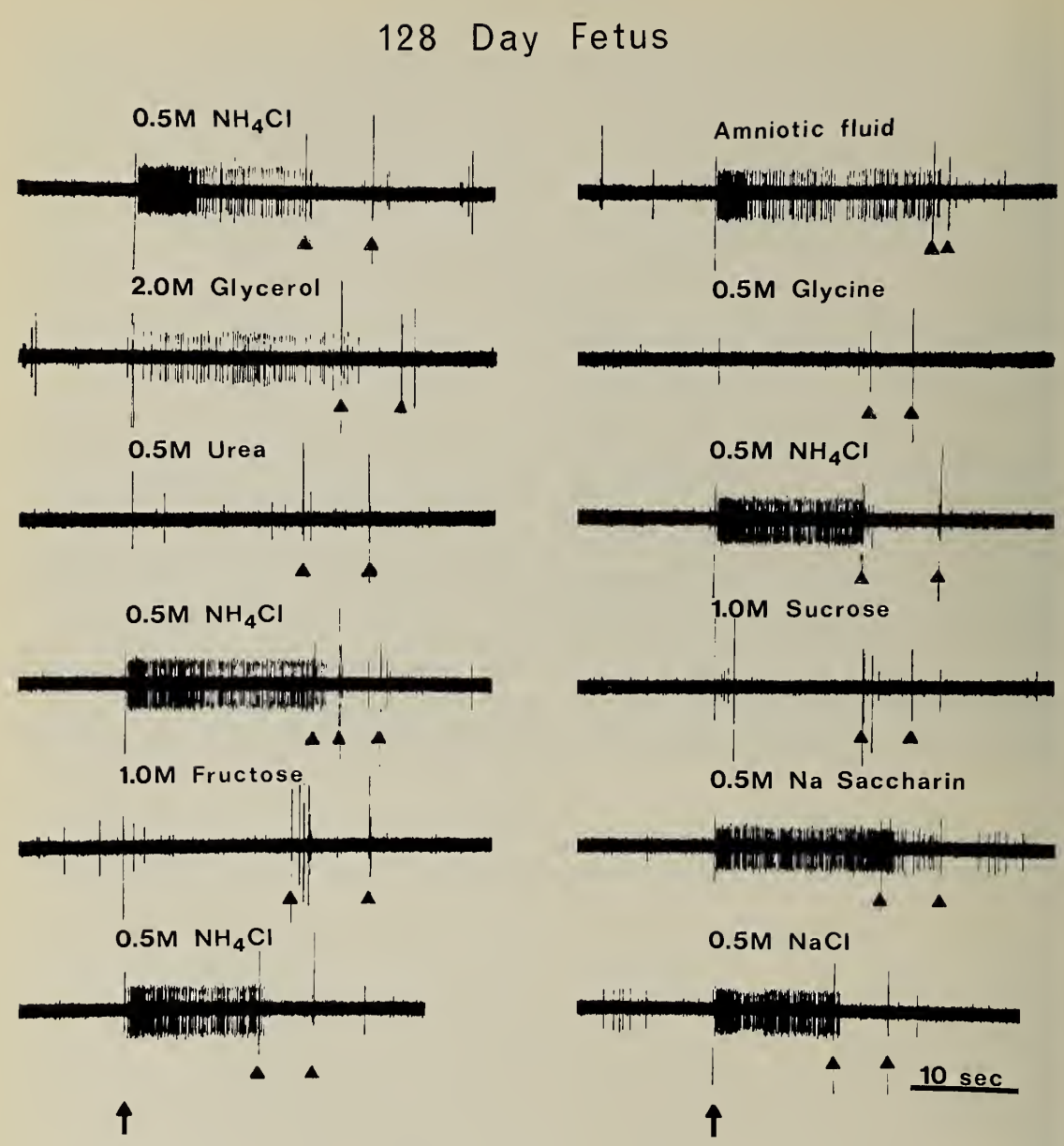


Figure 4–4. Neural impulses recorded from a single chorda tympani fiber in a 128-day fetus. Arrows at the bottom of each column denote time of stimulus application. Arrow heads in each trace indicate distilled water rinses. The record is continuous, and traces overlap, when read from top to bottom, left column, then right. (From Bradley and Mistretta.[7])

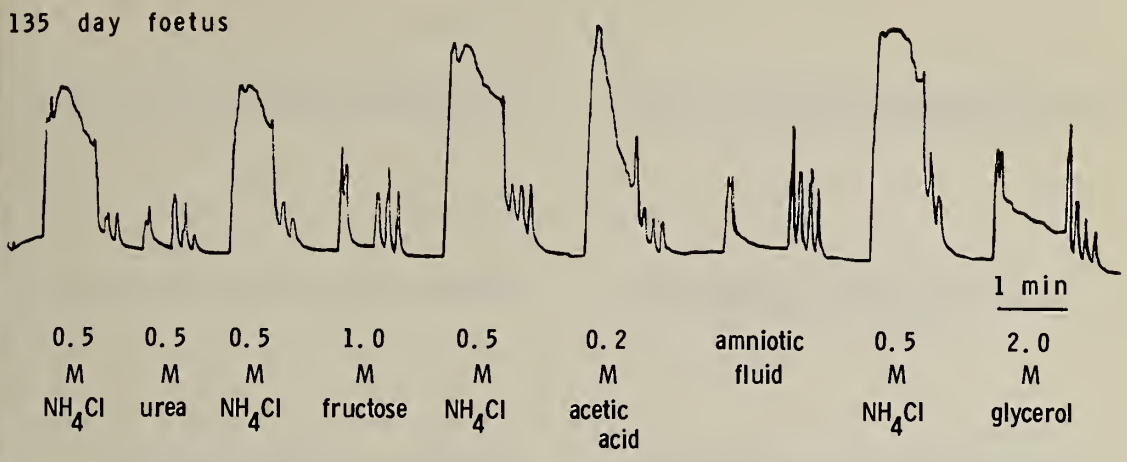


Figure 4–5. Integrated responses to a variety of stimuli recorded from a multi-fiber preparation in a 135-day fetus. (From Bradley and Mistretta.[7])

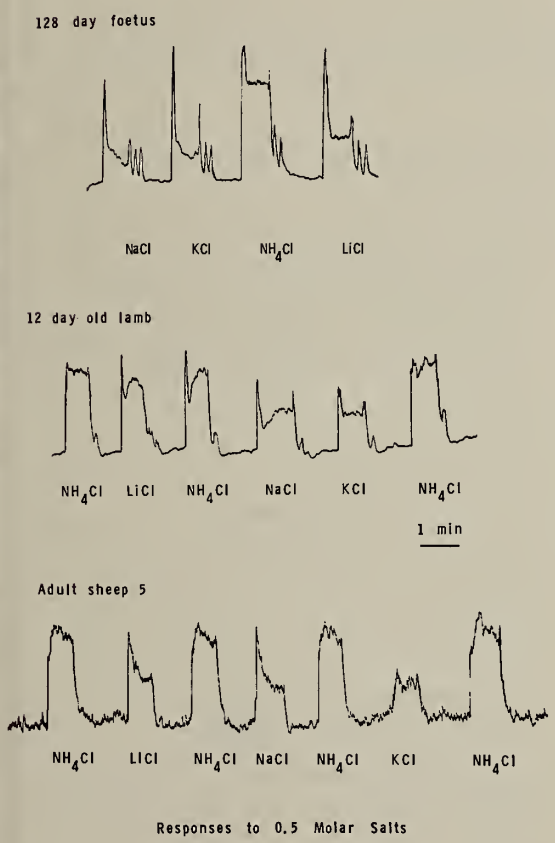

128 day foetus
NaCl KCl NH_4Cl LiCl
12 day old lamb
NH_4Cl LiCl NH_4Cl NaCl KCl NH_4Cl
1 min
Adult sheep 5
NH_4Cl LiCl NH_4Cl NaCl NH_4Cl KCl NH_4Cl
Responses to 0.5 Molar Salts

Figure 4–6. Integrated responses recorded from multi-fiber preparations during lingual stimulation with 0.5 M concentrations of monochloride salts. The order of effective salt stimulation is the same in these three preparations. (From Bradley and Mistretta.[7])

been obtained in fetuses aged 80 days to term. Most of the units in the region of the tractus solitarius respond similarly to those in the chorda tympani in that salts and acids elicit a high frequency neural discharge (Fig. 4–7). From experiments to date it seems that we will be able to use this technique to determine: (1) when taste buds are first responsive to chemical stimulation of the tongue, and (2) whether the neural response characteristics of the taste system change during development.

To summarize, it is known that human and sheep taste buds mature morphologically *in utero* long before birth. It is also known that sheep taste buds respond to chemical stimulation, at least from 80 days to term. But is the fetus itself actually behaviorally responsive to taste stimuli *in utero*? Can the fetus demonstrate preferences and aversions before birth? To answer these questions we have studied fetal swallowing activity and tried to experimentally alter that activity.

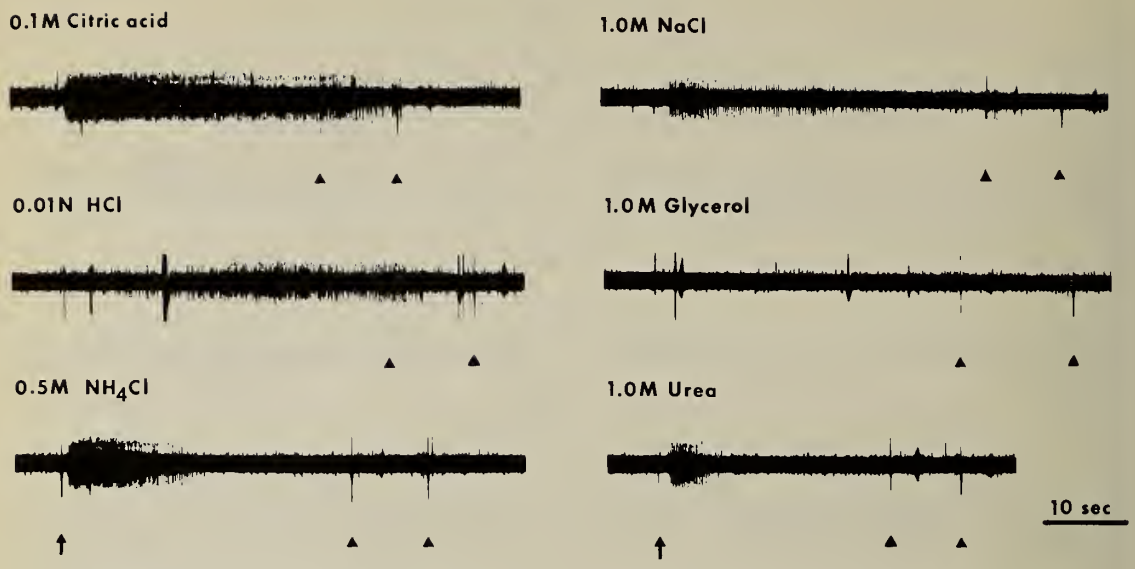


Figure 4–7. Neural responses recorded from units in the region of the tractus solitarius in a 140-day fetus. The arrows at the bottom of each column denote time of stimulus application. Arrow heads in each trace indicate distilled water rinses.

FETAL SWALLOWING

Swallowing has been demonstrated in many mammalian fetuses, including rats,[27] guinea pigs,[4] sheep,[8,9,32] and humans.[34] Although estimates of volume swallowed per 24 hours were available, there were no data on frequency of swallowing or volume swallowed at a given time. Using an electromagnetic flow transducer chronically implanted in the fetal esophagus we have been able to monitor fetal swallowing continuously for periods of weeks. Once baseline data on undisturbed swallowing were obtained, we then wished to modify swallowing. We hypothesized that if a pleasant-tasting chemical is injected into the amniotic fluid, and the fetus can behaviorally respond to taste stimuli, then the fetus should swallow more fluid after the intra-amniotic injection. Conversely, if an unpleasant-tasting chemical is injected, the fetus should swallow less after the intra-amniotic injection.

Thirty-eight dated pregnant ewes in the last third of gestation were operated under sterile conditions. The general surgical procedure is as described by Dawes et al.[17] The uterus was exposed with a midline incision, the fetal head palpated through the uterus and withdrawn through the abdominal incision. The uterus and fetal membranes were incised and the fetal head delivered. A cannulated electromagnetic flowmeter head [11] with an internal diameter of 3, 4 or 5 mm was inserted through a longitudinal incision into the fetal esophagus, and the incision closed. Catheters for monitoring fetal carotid and tracheal pressures were inserted into the carotid artery and trachea. Two catheters for sampling and injecting chemicals into the amniotic fluid were

sutured to the fetal head and snout. The fetal head was replaced in the uterus and uterine and abdominal incisions closed. The ewe was kept in a metabolism cage with food and water available *ad libitum*. Arterial and tracheal catheters were connected to pressure transducers and the externalized lead from the esophageal flow transducer was connected to a flowmeter. The flowmeter provided a continuous measurement of esophageal flow and, by integrating the flowmeter output, volume swallowed was monitored (Fig. 4–8). Esophageal flow rate, volume swallowed, and tracheal and arterial pressures were measured continuously, 24 hours per day, for periods of days and weeks.

Swallowing was monitored for 4 days or more in about half of the preparations. Volumes of amniotic fluid swallowed per day ranged from 50 to 785 ml. These large volumes of amniotic fluid swallowed by fetuses in the last third of gestation are comparable to those reported for human fetuses, ranging from 200 to 760 ml/day.[34] The fetus does not swallow on any obvious schedule, but at seemingly random times throughout the day (Fig. 4–9). Swallowing episodes are usually 1 to 9 minutes in duration. About 2 to 7 episodes occur per day. The volumes swallowed during an episode range from 20 to 200 ml and vary greatly for a given fetus on a given day.

Both the volume swallowed per episode and the total volume swallowed per day varied greatly for any fetus. The volumes swallowed per day by three fetuses are graphed in Figure 4–10. The daily volume usually peaks 3 to 5 days postoperatively, and falls off significantly as the ewe approaches labor. The variability of the timing of swallowing episodes, the volume swallowed per episode, and the volume swallowed per 24 hours, contributes to a very unstable baseline and makes it difficult to detect an effect of intra-amniotic chemical injections. When attempts to increase swallowing by injecting glucose into the amniotic fluid

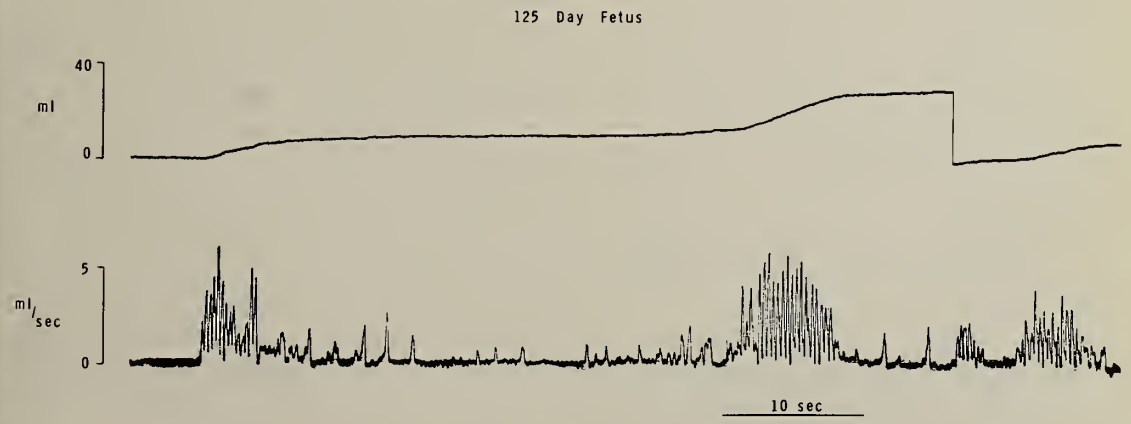


Figure 4–8. High speed record of swallowing activity from a 125-day fetus on the fourth postoperative day. The lower trace is the direct output of the electromagnetic flowmeter; positive deflections result from swallowing. The upper trace is the integrated flow and indicates volume swallowed. The integrator automatically resets to zero every 60 sec. (From Bradley and Mistretta.[8])

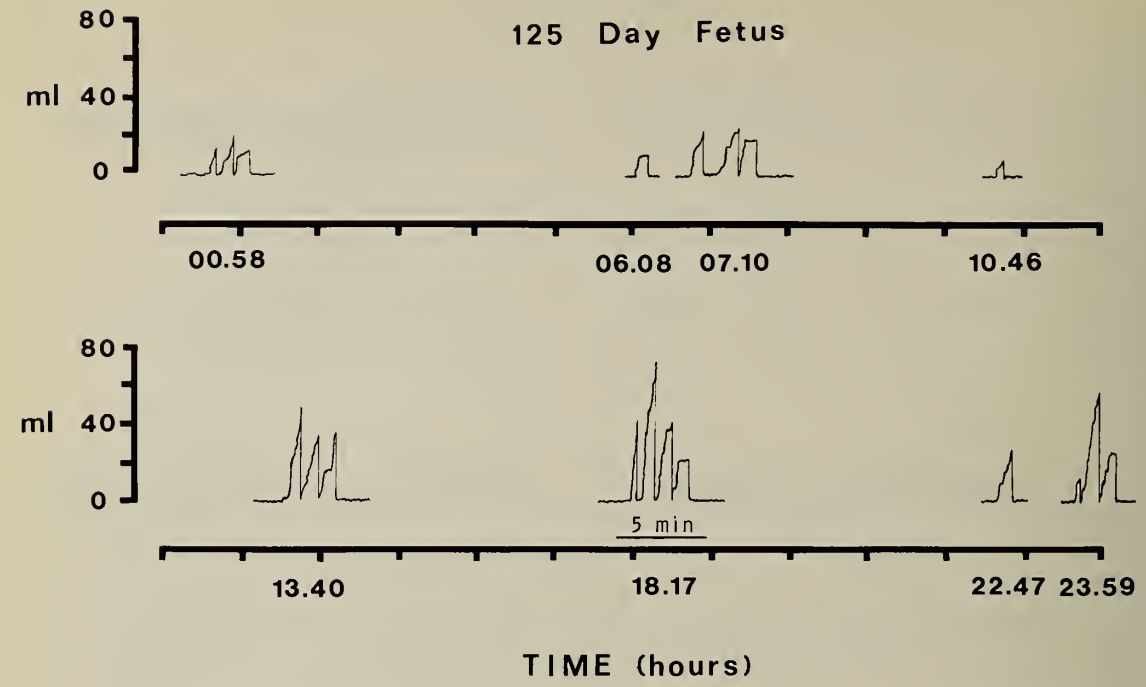


Figure 4–9. Integrated records of swallowing episodes over a 24-hour period from a 125-day fetus on the third postoperative day. The time at which swallowing occurred is indicated under each integrated recording. (From Bradley and Mistretta.[9])

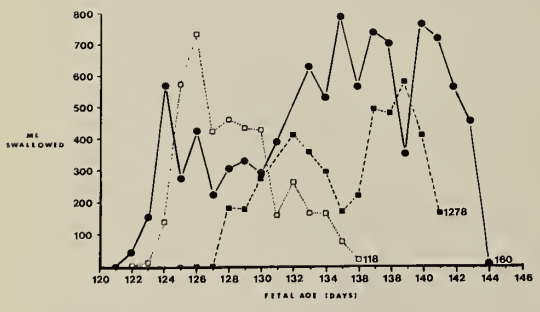


Figure 4–10. Volumes of amniotic fluid swallowed daily by three sheep fetuses. The data for each fetus are plotted from the day of operation for the duration of the experiment. No injections were made into the amniotic fluid of these fetuses. All ewes were in labor on the day of the last data point. (From Mistretta and Bradley.[32])

produced inconsistent results, we tried to decrease swallowing by injecting a bitter chemical, denatonium benzoate (Bitrex). Although in two fetal preparations we made repeated injections of Bitrex and the amniotic fluid on withdrawal tasted bitter to us, both fetuses continued to swallow large volumes of amniotic fluid. These experiments are still in progress.* Experiments by De Snoo [18] and Liley [29] suggest that the human fetus

* Recent work in our laboratory with M.L. Cheal suggests that denatonium benzoate is not aversive to newborn lambs. There is apparently a species difference between sheep and humans in the response to Bitrex. We are pursuing the swallowing experiments, using chemical which are aversive to new born lambs, such as quinine HCl and Na saccharin.

in the latter part of gestation responds to intra-amniotic injection of chemicals by swallowing more or less amniotic fluid over 24 hours. However, without adequate controls for viscosity and pressure effects these cannot be called taste responses.

From the experiments on fetal sheep, what conclusions can be made about the development of the sense of taste? First, the taste buds mature structurally long before birth, following a developmental sequence similar to that in the human. Secondly, for at least the last third of gestation in the sheep, the peripheral taste system is functional. Further, neurophysiological response characteristics over this period are similar to those of the adult. Thirdly, over the last third of gestation the fetus swallows large volumes of its liquid environment, the amniotic fluid. Although it is not yet possible to conclude that the fetus can actively respond to taste stimuli *in utero*, we hypothesize that the intrauterine taste experience may be biologically significant.

The human fetus swallows from about 12 weeks of gestation.[15] Taste receptors are present at this time, so for at least 28 weeks the gustatory system is exposed to chemicals in the amniotic fluid. Chemicals present in amniotic fluid include glucose, fructose, lactic, pyruvic and citric acids, fatty acids, phospholipids, creatinine, urea, uric acid, amino acids, polypeptides, proteins, and salts.[29] Since the chemical composition of amniotic fluid is not static, the receptors would not be adapted to a constant stimulus. The concentration of alanine in human amniotic fluid decreases from 0.65 mM at 8 weeks to 0.19 mM at term; valine decreases from 0.35 mM at 7 weeks to 0.07 mM at term.[28,37] Citric acid decreases from 0.3 mM at 6 months to 0.1 mM at term.[2] In the sheep Na^+ decreases from 116 mequiv/liter at 100 days of gestation to 86 mequiv/liter at term; over the same period urea increases from 6 mM to 12 mM.[30] These few examples illustrate the changing composition of amniotic fluid.

In addition to gradual changes in amniotic fluid composition, short term changes also occur. The fetus urinates into the amniotic fluid, the volume of urine ranging from 173 to 677 ml/day in the fetal sheep near term.[1,35] The sheep fetus urinates spontaneously throughout the day,[36] and the amniotic fluid composition would be altered after urination. In between swallowing episodes, when the fetus actually drinks amniotic fluid, the fetus' mouth is presumably closed. During this time outpourings of tracheal fluid [16] and possibly secretions from oral mucous and salivary glands may stimulate taste receptors.

The peripheral and central taste system develops in the presence of changing, low concentrations of a variety of chemicals. The intrauterine environment may influence the formation of neural connections in the taste system, just as the postnatal environment of the kitten influences the neural connections in the visual system.[23,24] Perhaps the prolonged fetal exposure to the low levels of chemicals in amniotic fluid contributes to neonatal rejection of high concentrations of chemicals or of chemicals not present *in utero*. The rat tends to associate novel chemical stimuli (e.g., those which do not taste familiar) with poison more readily than familiar

stimuli.[25] The newborn may similarly reject novel chemical stimuli. One would predict, then, that newborns should prefer chemicals at concentrations close to those in amniotic fluid over chemicals at widely differing concentrations or over chemicals not present in amniotic fluid.

Imposed on the prenatal taste experience is, of course, the postnatal exposure to taste stimuli discussed at the beginning of this paper. The postnatal taste experience in the human comes at the time of major development of neural connections in the cerebral cortex.[12,13,14] In species born in a precocial state, however, such as the sheep and guinea pig, more cortical development takes place before birth.[3] So for these species the prenatal taste experience may have a greater influence on the gustatory system.

Although we do not yet understand the significance of the early, intrauterine structural and functional development of the taste receptors, we do have enough information to suggest that prenatal experience cannot be ignored. When studying the interaction of genetic and environmental factors in the acquisition of taste preference and aversion, it is not valid to conclude that the presence of a preference at birth demonstrates a genetic basis. Much experience with taste stimuli in the amniotic fluid environment has already taken place.

REFERENCES

1. Alexander, D.P., Nixon, D.A., Widdas, W.F., and Wohlzogen, F.X.: Renal function in the sheep foetus. *J. Physiol.*, 140:14–22, 1958.
2. Anteby, S.C., Zukerman, H., Gedelia, 1., and Weistreich, V.: Citric acid in amniotic fluid. *J. Obstet. Gynaec. Brit. Comm.*, 80:27–28, 1973.
3. Astrom, K.E.: On the early development of the isocortex in fetal sheep. In Bernhard, C.G. and Schadé, J.P. (Eds.), *Progress in Brain Research, Vol. 26, Developmental Neurology,* New York, Elsevier, 1967, pp. 1–59.
4. Becker, R.R., Windle, W.F., Barth, E.E., and Schulz, M.D.: Fetal swallowing, gastro-intestinal activity and defecation in amnio. *Surg. Gynecol. Obstet.*, 70:603–614, 1940.
5. Beidler, L.M.: Properties of chemoreceptors of tongue of rat. *J. Neurophysiol.*, 16:595–607, 1953.
6. Bradley, R.M.: Development of the taste bud and gustatory papillae in human fetuses. In Bosma, J.F. (Ed.), *The Third Symposium on Oral Sensation and Perception: The Mouth of the Infant,* Springfield, Thomas, 1972, pp. 137–162.
7. Bradley, R.M. and Mistretta, C.M.: The gustatory sense in foetal sheep during the last third of gestation. *J. Physiol.*, 231:271–282, 1973.
8. Bradley, R.M. and Mistretta, C.M.: The sense of taste and swallowing activity in fetal sheep. In Comline, R.S., Cross, K.W., Dawes, G.S. and Nathanielsz, P.W. (Eds.), *Foetal and Neonatal Physiology. Proceedings of the Sir Joseph Barcroft Centenary Symposium.* Cambridge, Cambridge Univ. Press, 1973, pp. 77–81.
9. Bradley, R.M., and Mistretta, C.M.: Swallowing in fetal sheep. *Science,* 179:1016–1017, 1973.
10. Bradley, R.M., and Stern, I.B.: The development of the human taste bud during the foetal period. *J. Anat.*, 101:743–752, 1967.
11. Clark, D.M. and Wyatt, D.G.: An improved perivascular electromagnetic flowmeter. *Med. Electron. and Biol. Engng.*, 7:185–190, 1969.
12. Conel, J.L.: *The Post-natal Development of the Human Cerebral Cortex. I. Cortex of the Newborn,* Cambridge, Harvard Univ. Press, 1939.
13. Conel, J.L.: *The Post-natal Development of*

the Human Cerebral Cortex. II. Cortex of the One-month Infant, C ambridge, Harvard Univ. Press, 1941.

14. Conel, J.L.: *The Post-natal Development of the Human Cerebral Cortex. III. Cortex of the Three-month Infant,* Cambridge, Harvard Univ. Press, 1947.
15. Davis, M.E., and Potter, E.L.: Intrauterine respiration of the human fetus. *J.A.M.A.,* 131:1194–1201, 1946.
16. Dawes, G.S.: Breathing and rapid-eye-movement sleep before birth. In Comline, R.S., Cross, K.W. Dawes, G.S. and Nathanielsz, P.W. (Eds.), *Foetal and Neonatal Phsyiology. Proceedings of the Sir Joseph Barcroft Centenary Symposium,* Cambridge, Cambridge Univ. Press, 1973, pp. 49–62.
17. Dawes, G.S., Fox, H.E., Leduc, B.M., Liggins, G.C., and Richards, R.T.: Respiratory movements and rapid eye movement sleep in the foetal lamb. *J. Physiol.,* 220:119–143, 1972.
18. De Snoo, K.: Das trinkende Kind im Uterus. *Monatsschr. Geburtsh. Gynaekol.,* 105:88–97, 1937.
19. Desor, J.A., Maller, O., and Turner, R.E.: Taste in acceptance of sugars by human infants. *J. Comp. Physiol. Psych.,* 84:496–501, 1973.
20. Dews, P.B., and Wiesel, T.N.: Consequences of monocular deprivation on visual behaviour in kittens. *J. Physiol.,* 206:437–455, 1970.
21. Farbman, A. I.: Electron miscroscope study of the developing taste bud in rat fungiform papilla. *Dev. Biol.,* 11:110–135, 1965.
22. Galef, B.G., and Sherry, D.F.: Mother's milk: a medium for transmission of cues reflecting the flavor of mother's diet. *J. Comp. Physiol. Psych.,* 83:374–378, 1973.
23. Hubel, D.H. and Wiesel, T.N.: Binocular interaction in striate cortex of kittens reared with artificial squint. *J. Neurophysiol.,* 28:1041–1059, 1965.
24. Hubel, D.H., and Wiesel, T.N.: The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *J. Physiol.,* 206:419–436, 1970.
25. Kalat, J.W.: Taste salience depends on novelty, not concentration, in taste-aversion learning in the rat. *J. Comp. Physiol. Psych.,* 86:47–50, 1974.
26. Le Magnen, J., and Tallon, S.: Préférence alimentaire du jeune rat induite par l'allaitement maternel. *C. R. Soc. Biol.,* 162:387–390, 1968.
27. Lev, R., and Orlic, D.: Protein absorption by the intestine of the fetal rat in utero. *Science,* 177:522–524, 1972.
28. Levy, H.L., Easterday, C.L., Montag, P.P., and Littlefield, J.W.: Amino acids in amniotic fluid. In Dorfman, A. (Ed.) *Antenatal Diagnosis,* Chicago, Chicago Univ. Press, 1970, pp. 109–113.
29. Liley, A. W.: Disorders of amniotic fluid. In Assali, N. S., *Patho-physiology of Gestation, Vol. II. Fetal-Placental Disorders.* New York, Academic, 1972, pp. 157–206.
30. Mellor, D.J. and Slater, J.S.: Daily changes in amniotic and allantoic fluid during the last three months of pregnancy in conscious, unstressed ewes with catheters in their foetal fluid sacs. *J. Physiol.,* 217:573–604, 1971.
31. Mistretta, C.M.: Topographical and histological study of the developing rat tongue, palate and taste buds. In Bosma, J.F., (Ed.), *The Third Symposium on Oral Sensation and Perception: The Mouth of the Infant,* Springfield, Thomas, 1972, pp. 163–187.
32. Mistretta, C.M., and Bradley, R.M.: Taste and swallowing *in utero:* a discussion of fetal sensory function. *Brit. Med. Bull.,* 31:80–84, 1975.
33. Nachman, M.: The inheritance of saccharin preference. *J. Comp. Physiol. Psych.,* 52:451–457, 1959.
34. Pritchard, J.A.: Deglutition by normal and anencephalic fetuses. *Obstet. Gynecol.,* 25:289–297, 1965.
35. Rankin, J.H.G., Gresham, E.L., Battaglia, F.C., Makowski, E.L., and Meschia, G.: Measurement of fetal renal inulin clearance in the chronic sheep preparation. *J. Appl. Physiol.,* 32:129–133, 1972.
36. Robillard, J.E., Thayer, K., Kulvinskas, C., and Smith, F.G.: Spontaneous activity of the fetal bladder. *Am. J. Obstet. Gynecol.,* 118:548–551, 1974.

37. Scott, C.R., Teng, C.C., Sagerson, R.N., and Nelson, T.: Amino acids in amniotic fluid: Changes in concentrations during the first half of pregnancy. *Pediat. Res.*, 6:659–663, 1972.
38. Stockton, M.D., and Whitney, G.: Effects of genotype, sugar, and concentration on sugar preference of laboratory mice (*Mus musculus*). *J. Comp. Physiol. Psych.*, 86:62–68, 1974.
39. Warren, R.P., and Pfaffmann, C.: Early experience and taste aversion. *J. Comp. Physiol. Psych.*, 52:263–266, 1959.

DISCUSSION

STANLEY: Have you considered recording tongue responses?

MISTRETTA: No. We have not recorded any fetal movements.

STANLEY: Do you think that the rate of introduction of stimuli was so slow that it didn't provide an effective stimulus?

MISTRETTA: It may be that rapidly injecting a large volume of a more concentrated stimulus would be more effective in eliciting a response. We have tried two methods of introducing stimuli into the amniotic fluid. Usually we inject into a catheter that is sutured to the fetal head. When stimuli are injected, they flow down over the nose. In a few preparations, we sutured a catheter to the inside of the fetal cheek; stimuli were actually introduced intra-orally. Neither method produced consistent results.

We have an additional problem to consider in these experiments—that is the question of the "state" of the fetus. We may hear more about this tomorrow from Dr. Johnson in relation to his studies on newborns. The point is that fetuses are only awake and responsive a certain portion of the time. If we inject a stimulus during an unresponsive period, we cannot expect to induce swallowing.

JOHNSON: Just on this point of the state of the fetus, I think there is some information which would support your concern about the state of the fetus and the species difference. It is clear now that the sheep's fetal breathing movements occur periodically and its sleep state is cyclical and well defined. It is also clear one can do very little to stimulate or modify the breathing in a non-breathing period, so that nearly 50 to 60 percent of the time there is nothing we can do to either promote or depress breathing, in the fetal lamb. During the other periods, when the fetus is breathing, we know that we can modify that breathing. At these times the fetus is extremely sensitive to respiratory stimuli. The same may apply to swallowing activity.

In terms of Liley's work on human fetuses, the relative sleep periods are quite different from those in sheep. This might explain the differences in experimental results.

MISTRETTA: We may be forced to monitor the EEG in our fetuses, as well as other parameters.

COLLIER: I would like to suggest a far-fetched interpretation of the results. If you look at guinea pigs and the frequency of the feeding bouts that they have from the day they are born until they are a year or two old, you find they feed about 22 times a day. This is very constant even though they are growing and the amount they eat is changing very greatly. Perhaps what we have is a predetermined frequency of feeding that is genetically

determined, and I am wondering if the bouts of fetal swallowing in your animals might just have the same frequency as the feeding of these animals in the natural state under normal circumstances. Then what you are looking at is the predetermined frequency of feeding rather than anything to do with taste.

MISTRETTA: I agree that fetal swallowing could be the first manifestation of mammalian feeding behavior. It may be that individual variations in the frequency of fetal swallowing bouts per day are the first indication of a preferred newborn feeding schedule. But I don't see why this should preclude the possibility of inducing the fetus to drink more or less per bout depending on the "flavor" of the amniotic fluid.

LIPSITT: We know from studies of the neonate, both human and canine, that the most effective stimulus for eliciting sucking is an intra-oral stimulus shaped as closely as possible like that of the natural mother. I would like, therefore, to make the suggestion, without being able to be entirely specific, that you have to find some way of putting a nipple into the mouth of the fetus in utero in order to take the best possible look at differential taste responses. After all, the way to optimize sucking in the first place is to use an appropriately shaped intra-oral stimulus. Taste controls the sucking parameters after touch starts the process. You have to have the right shape nipple to start sucking, then the right taste to keep it going.

WEIFFENBACH: Are you suggesting that taste may be capable of modifying sucking but that it does not so readily modify swallowing?

LIPSITT: I did not have that in mind. This might be a worthwhile hypothesis. My own view would be that both sucking and swallowing are modified by taste, but we are not clear on the relationships at the present time. This is an area of investigation that is still wide open. I suggest that if a suitable means of offering the fetus an optimizing intra-oral stimulus through which very small amounts of tastants could be delivered, both sweet and bitter, the fetus' sucking movements would provide excellent clues as to the organism's affinity for these substances.

MISTRETTA: Technically I don't know if it will be possible to do what you suggest, but you've given us something to think about.

PFAFFMANN: It occurs to me with the time rise and fall of the stimulus you use, there is the likelihood of adaptation or habituation. In your foetal sheep when the taste buds are formed, the amniotic medium would seem to elicit a continuous barrage of activity all the time and to which you either add or subtract stimulation. I think you would have to devise a way of producing a more abrupt impact of impulses, perhaps by flushing out the oral cavity with water or some other neutral solution.

MISTRETTA: To produce a more abrupt stimulation we tried injecting stimuli directly into the fetal mouth, as I mentioned to Dr. Stanley. This did not improve the likelihood of inducing swallowing.

NOWLIS: I have a few thoughts that might be relevant to a pursuit of fetal swallowing. Miller and Sherrington [1]

[1] Miller, F. R., and Sherrington, C.S. Some observations on the buccopharyngeal stage of reflex deglutition in the cat. *Quart. J. exper. Physiol.,* 9:147–186, 1916.

found in the acute decerebrate cat that almost any fluid, noxious or otherwise, would elicit a swallow when administered to the wall of the pharynx, the soft palate, the epiglottis, or the extreme posterior portion of the dorsum of the tongue. Most noxious fluids, including quinine, seemed not to have an inhibitory influence on the swallow on the first few applications, but subsequent applications elicited not swallowing, but retching. This is a very interesting finding. The swallow is important enough in keeping fluids out of the airways that apparently its elicitation is not inhibited unless another reflex which can protect the airways has been recruited. Whether this inhibition of the swallow is a function of direct inhibition from the retch reflex to the noxious substance in the stomach, or a conditioned retch now elicitable with direct chemical stimulation of the oropharynx is an interesting empirical question. In either case, it seems possible that the maturation of this relationship between retch and swallow could be a prerequisite for negative gustatory influence over the swallow.

In the case of influence from the taste buds on the anterior tongue, another type of reflex [1] may be necessary to affect the likelihood of a swallow, whereby the tongue either pushes a bolus back toward the receptive field for swallow, or out of the mouth, depending on the taste. Apparently one has to wait a week or two after decerebration [2,3] before such reflexes appear. It would be interesting to know whether such tongue movements are present in your fetal preparation, and if so, whether they can produce any local fluid pressure fluxes in the area of the receptive field for swallow. Since this area is already replete with fluid in the fetus, it would be exciting simply to find what it is that elicits swallowing in this preparation.

MISTRETTA: Thank you for your interesting and informative comments. I think that one thing to keep in mind, though, is that you could look on our experiments as fetal preference tests. That is, whether or not we "turn on" a swallow may not be of significance. But rather, if the amniotic fluid is constantly modified to taste unpleasant, will the fetus on beginning to swallow, stop swallowing or swallow less because it detects something aversive? And vice versa for pleasant tasting injections?

WEIFFENBACH: I think there have been several of us saying the same thing. At least, I hope the other people are saying what I am trying to say. Consider a model in which taste stimulation of the tongue affects responding in the muscles that control the shape of the mouth, that is sucking. These sensory motor connections might or might not be functional, but without a nipple to complete the seal at mouth there might be no effect on swallow. Beyond the relation of taste stimuli on the tongue to mouth shape

[1] Nowlis, G.H. Taste-elicited tongue movements in human newborn infants: An approach to palatability. In: Bosma, J.F. (Ed.), *Fourth Symposium on Oral Sensation and Perception,* Washington, Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 272–303.

[2] Bard, P. and Macht, M. B. The behaviour of chronically decerebrate cats. In: Wolstenholme, G.E.W., (Ed.) *Ciba Foundation Symposium on the Neurological Basis of Behavior.* Boston: Little, Brown, 1958, 55–75.

[3] Macht, M.B. Subcortical localization of certain "taste" responses in the cat. *Fed. Proc.,* 1951, 10: 88 (abstract).

there is the relation of fluid pressures to swallowing. Even if the taste-suck mechanism were capable of performance from the time the taste buds became functional, the mechanical arrangement of the mouth might keep the taste related changes in shape from leading to swallow until the organism prehends a nipple and thus alters the relation between changes in mouth shape and pressure.

One consequence of this is, I believe, that you might think of seeing if the taste stimuli you deliver elicit any non-swallow feeding movements.

DESOR: Do your esophogeal flowmeters measure flow in both directions?

MISTRETTA: Yes.

DESOR: Have you observed any regurgitation? I'm asking because we have observed some regurgitation of sour stimuli by human newborns.[1] The infants did not reduce their intake of water when a sour compound was added to it, but after ingestion, the sour solution was more likely to come back up. This suggests the possibility that food rejection in newborns involves not only the oral cavity, but the gastrointestinal tract down to and including the stomach. Clearly, newborns exert some control at the oral level (e.g., they take in less water than sweet solutions [2]), but there may be a further screening of fluids somewhere further down the GI tract. This would be adaptive since it appears that the newborn cannot completely inhibit ingestion: at least some small amount of anything offered through a nipple to a newborn seems to get to his stomach.[1]

There is an interesting extension of this observation suggested by discussions with Paul Rozin. First, it seems to me that infants do alot of gagging and vomiting in general. Anyone who has been around infants will probably agree that it is not uncommon to have them spit up on you, whereas it is unusual for toddlers or adults to regurgitate their food. Second, if you think about how older infants or toddlers reject food, it seems they often take food (and other things) into the mouth and then spit them out. Perhaps at this age, there is little inhibition of taking-into-the-mouth, but swallowing can be inhibited, so that at this stage, some amount of nearly everything offered reaches the oral cavity and is rejectable from there. Adults typically reject food *outside* the mouth; selection is made prior to putting anything in the mouth. In adults, virtually anything that is taken into the mouth is committed to going through the entire GI system. Neither spitting out (oral rejection) nor regurgitation (stomach rejection) is a noticeable part of the adult repetoire.

These informal observations suggest a developmental sequence of the rejection side of food selection which complements Dr. Nowlis' theory (Chap. 14). It may be that early in development, some amount of anything offered reaches the stomach and is easily rejectable from there. With development, the "point of no return" moves progressively forward such that in toddlers, it is inside the mouth and in adults, it is outside the mouth.

MISTRETTA: We have not observed

[1] Desor, J., Maller, O., & Andrews, K. Ingestive responses of human newborns to salty, sour, and bitter stimuli. *Journal of Comparative and Physiological Psychology*, in press.

[2] Desor, J., Maller, O., & Turner, R. Taste in acceptance of sugars by human infants. *Journal of Comparative and Physiological Psychology*, 1973, 84:496–501.

significant movement of fluid out of the esophagus, that is, from stomach to mouth. The sheep does not begin to ruminate until some weeks after birth, so we did not expect to find evidence of rumination.

WEBBER: Since you have essentially a closed system in utero, it seems to me it is pretty difficult to ignore food balance in general as a mediating influence over the long run in terms of the amount of fluid you have taken in.

MISTRETTA: At the Nuffield Institute for Medical Research in Oxford we were fortunate to collaborate for a short time with Dr. Heather Shelley. We were injecting glucose into the amniotic fluid, assuming 1000 ml of amniotic fluid and trying to attain a concentration of one molar, which is very high. Dr. Shelley found that the glucose levels in the amniotic fluid had returned to normal 18–24 hours post-injection. We have not been able to monitor disappearance of Bitrex but have tasted the amniotic fluid every day after injection. The bitter taste remains for 3–4 days.

GRINKER: I wonder whether the bouts of foetal swallowing can be related to the mother's short-term nutritional status. Is there a relationship between the mother's feeding and the swallowing?

MISTRETTA: We haven't done any experiments on possible relationships between the ewe's nutrition and fetal swallowing. Food and water are available to the ewe *ad libitum.*

GALEF: It's not clear to me on what model you predict a different rate of swallowing in fetal sheep as a function of the taste of the amniotic fluid. Because the sensory receptors are continuously bathed in the fluid regardless of the behavior of the sheep, there are no reinforcement contingencies resulting from active swallowing. There is not much reason to expect changes in frequency of swallowing on an operant model.

MISTRETTA: The reason to expect more swallowing is that if the fetus can detect a pleasant tasting fluid, it should choose to ingest more of it; if it detects something unpleasant, it should ingest less of it, or none at all. The reinforcement would be provided by the taste of what the fetus drinks.

GALEF: Only assuming that the swallowing, like sucking, turns out to be under stimulus control by taste. There doesn't seem to be good reason to wire the animal that way because in the normal circumstances the amniotic fluid remains relatively constant. There aren't going to be tastes there.

MISTRETTA: I cannot agree that there are not taste stimuli normally present in amniotic fluid. The concentrations of various chemicals in amniotic fluid are changing daily. After fetal urination, the chemical composition also changes. Although we do not know whether the fetus can detect these changes, we cannot yet rule out of the possibility that taste stimuli are usually present *in utero.*

PFAFFMANN: A number of speakers have been remarking that sucking is under the stimulus control of taste. I would prefer to believe that but, as I read the literature, it doesn't appear to be the case. Sucking is under the primary control of tactual or mechanical pressure. Taste can be shown to have an effect once sucking gets going.

In evolutionary terms, why should some of these tastes be present at the neonatal stage in so many mammals?

One could say they are there to insure that the animal maintains the sucking reflexes once started. Sucking may be sweet-dependent or fat-dependent or dependent on many other things that are in the milk. But the primary stimulus is tactual—as I think Walt Stanley pointed out. If you put your finger in the mouth of some of the young beagle pups, they will hang onto your finger. Sucking is a very strong response regardless of whether the pups are getting anything or not, although I know there will be evidence presented here that shows taste to have an important modulating influence.

MISTRETTA: But once an animal is drinking, or once a fetus is swallowing, taste can certainly modify ingestion.

WEIFFENBACH: It would seem we have a two-stage modulation. In order for a tactual stimulus to be a good eliciter of sucking, you have to have an infant in the appropriate state. Then, given that you have an organism that is in a state to suck and that he is sucking, the potential exists for using taste as a modifier of that ongoing sucking.

Chapter 5

DEVELOPMENTAL CHANGES IN RAT FUNGIFORM PAPILLA POPULATIONS

Patricia W. Henderson and Grant K. Smith

Taste buds depend on an intact nerve supply for their development and continued existence. They disappear following denervation of the tongue [27,26,19–21,14,28,23,24,12,13,3,29–34] and regenerate following reinnervation by a gustatory nerve.[19,20,13,29–34] Attempts to induce regeneration *in situ* by reinnervation with non-gustatory sensory or motor nerve have not been successful[13,31,18] although explants of lumbar sensory ganglia have induced taste bud formation in vallate papillae grafted into the anterior chamber of the eye.[34]

These and similar observations have been used by analogy to explain the processes underlying ontogenetic development and the analogy has received some support. For example Torrey [25] pointed out that although the gustatory nerve enters the epithelium of the rat tongue about the day of birth, taste buds do not develop until about the ninth day of life. In a series of experiments also employing transplants into the anterior chamber of the rat eye, he showed that taste buds will develop in the absence of nerve provided that the transplant is done between days 7 and 9 postpartum. The results indicate that on or about day 7 the nerve exerts a trophic effect on the epithelium which induces it to differentiate into taste buds on day 9 even though the nerve is decentralized.

The mechanisms responsible for the localization of the gustatory nerve and the development of taste buds in gustatory epithelium are much less clear. Taste buds occur mainly on the dorsal surface of the tongue in association with papillae of some sort although they are also found on the lips, nasopharynx and other portions of the buccal mucosa not in association with papillae.[15] An identifiable, differentiated anatomical structure is, apparently, not necessary for the formation of taste buds. When the papilla is present, however, it seems to be the precursor of taste bud formation as its development begins before the nerve penetrates the epithelial layer.[7] In the adult rat testosterone treatment can cause taste buds to develop on areas of the circumvallate papilla where they do not normally occur, but not on other areas of the tongue.[30] Farbman [7] reports that children suffering from familial dysautonomia, a syndrome which includes disturbances of taste perception and discrimination, seem completely lacking in fungiform papillae. He is led to conclude that papillae provide loci on the dorsum of the tongue with a predisposition for the development of taste buds. It appears

that papillae may create the conditions for growth of the nerve into proximity with cells of the papillary epithelium which then differentiate into taste buds under the trophic influence of the nerve. The stimulus for the formation of papillae is not known.

Thus taste bud bearing papillae of the rat tongue seem to be deeply involved in determining the location of taste bud development. There is evidence also that they may strongly influence the response characteristics of gustatory nerve fibres. Oakley [17] describes cross-innervation experiments in which the proximal end of the chorda tympani is grafted into the distal stump of the glossopharyngeal nerve. Following regeneration, the pattern of sensitivity to sapid stimuli of fibres of the chorda tympani nerve changes to that characteristic of glossopharyngeal fibres. In view of this evidence that the papillae may play a dominant role in the development of gustatory receptors we have undertaken a study of the course of development of the papilla population and factors which may influence this development.

The taste bud bearing papillae of mammalian tongue fall into three classes–circumvallate which lie along a V-shaped ridge dividing the tongue, foliate usually on the lateral margins of the tongue, and fungiform on the anterior two thirds. Filiform papillae are usually rough projections of cornified epithelium which cover the anterior two thirds of the tongue and do not carry taste buds. In the adult rat the fungiform papillae each bear a single, central taste bud. Papillae begin to develop prenatally, and are visible on the surface of the tongue about the time of birth. Over the first two to three weeks of extrauterine life innervation occurs and taste buds develop.[25,7] It is during this same period that the animal changes its mode of food intake twice, and develops taste preferences dependent on the maternal diet which carry forward into adulthood.[9] It seemed reasonable to speculate that, among the unknown factors determining the pattern of papilla (and hence taste bud) development, there may be some components of the early feeding experience.

There are reports in the literature that more taste buds may be found on the tongues of foetal and infant rats than on those of adults.[6,2] These claims have not been accompanied by reports of differences in the number of *papillae* on infant and adult tongues.[8] The work we will describe first examines in detail the number of fungiform papillae on the rat tongue from before birth to about 3 months of age. It then attempts to isolate some of the variables that may be responsible for determining the timing of the development of the papilla population.

Long-Evans hooded rats were bred in the laboratory. Pregnancies were dated from the first appearance of sperm in the vaginal smear. Some foetuses were taken by caesarian section under barbiturate anaesthesia, others were born normally and housed with their biological mothers or cross-fostered as described below. Tongues were removed under barbiturate anaesthesia and the surface was stained with methylene blue. Fungiform papillae do not take up the dye and are seen in contrast to filiform papillae which stain dark

blue and surrounding epithelium which stains less darkly. The number and distribution of papillae on each tongue were obtained from large scale maps drawn with the aid of a binocular operating microscope. Each data point is the mean of counts from four animals of the same age but from different litters. The confidence limits indicated on the graphs are the standard deviations.

EXPERIMENT 1: THE NORMAL POPULATION GROWTH CURVE

Methods

Samples were taken just before birth and at five day intervals thereafter to 90 days. The first eight hours of extrauterine life and the period between days 15 and 25 were then investigated in more detail. Up to day 35 the animals were housed with their biological mothers in separate laboratory cages, with food and water available, and allowed to wean normally. Following separation from the mother two animals of the same sex were housed in each cage.

Results

The results are plotted in Figure 5–1 and show that on the 20th day of gestation (birth normally occurs on the 21st) no fungiform papillae can be seen by our method. By the fifth postnatal day the number has risen to

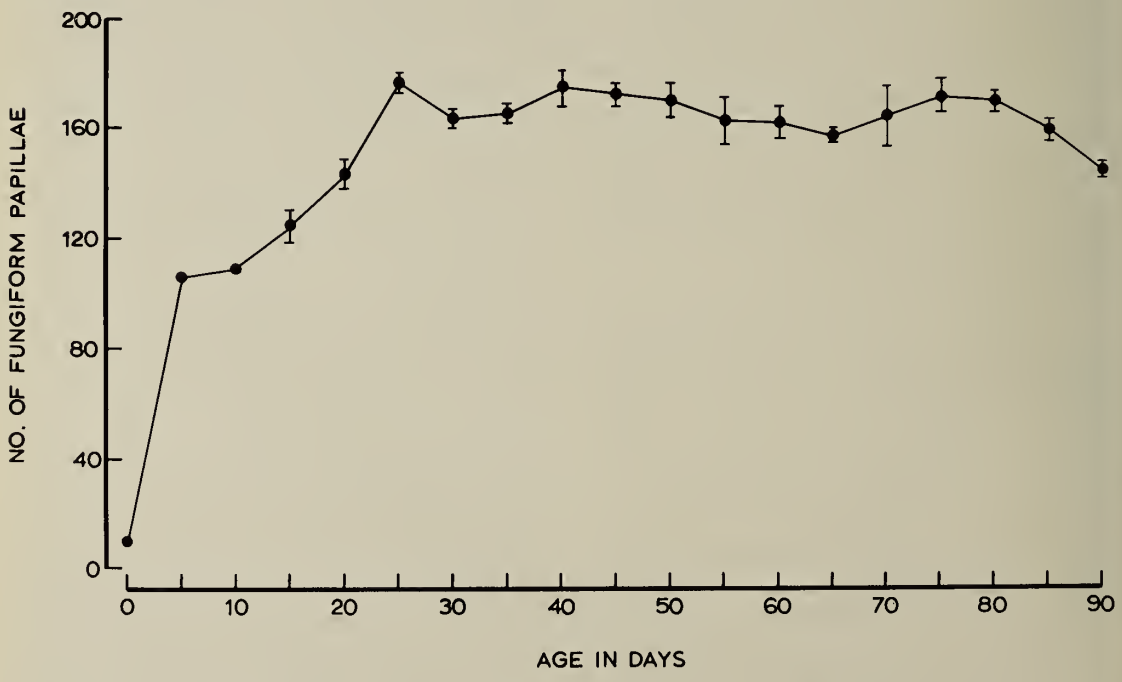


Figure 5–1. The curve of numbers of non-staining fungiform papillae visible on the tongues of hooded rats through the first 90 days. There are two postnatal periods of increase, the first (Phase 1) occurring by day 5, the second (Phase 2) between days 15 and 25.

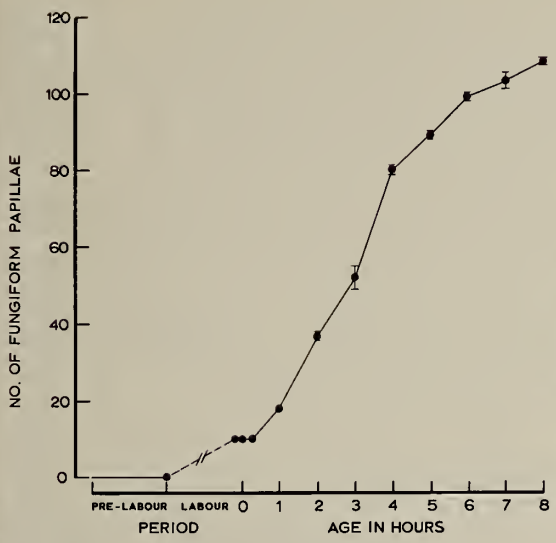


Figure 5–2. Results of a detailed study of phase 1. Non-staining papillae first appear during labor and their numbers increase rapidly during the first 4 postnatal hours, the rate of increase then slows. Phase 1 increases are essentially complete by the end of the eighth postnatal hour.

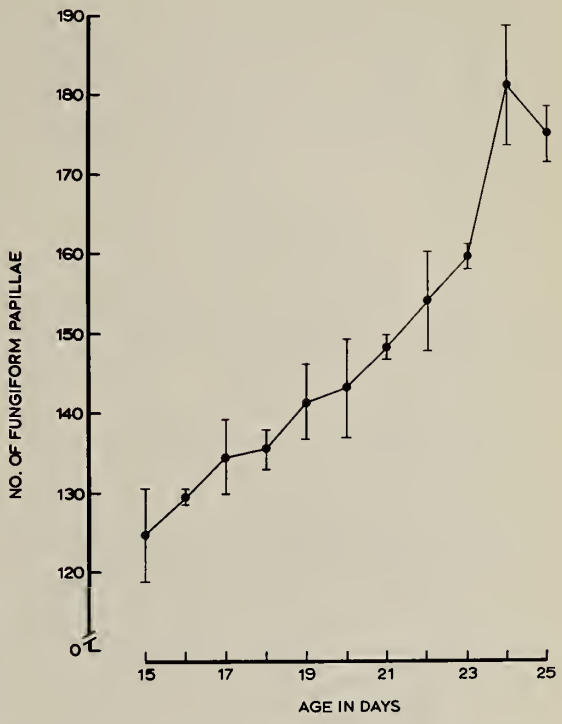


Figure 5–3 Phase 2 of the papilla population curve. This development takes place over the 3rd and 4th week of postnatal life, coinciding with the time of weaning.

about 110 where it remains virtually unchanged through day 10. By day 15 the number has begun to increase again and it continues to rise until day 25 when a second plateau is reached at about 170; around day 80 the number declines again slightly to the adult level of about 150.

A closer examination of the two growth phases was then made. Daily samples over the first five postnatal days indicated that the initial growth phase (Phase 1) takes place before the neonate is 24 hours old, as all counts over this period were approximately 110. Foetal rats were examined on the 20th day of gestation, and there were no fungiform papillae discernible on the tongues at this stage of development. On the 21st day of gestation, after the onset of labour, five pairs of papillae appear arranged along the central sulcus in the mid-portion of the tongue (Fig. 5–4). Shortly after birth a rapid growth period begins, (Fig. 5–2) slowing after 4 hours and ending after 8 hours. There is very little variability among tongues of the same age.

Daily samples were also taken from day 15 to day 25 and the results are shown in Figure 5–3. This growth period (Phase 2) is much slower than the first one, more uniform in the rate of change, but variability among animals of the same age is somewhat greater.

Figure 5–4 shows maps of four tongues made on the day of birth indicating the pattern of development

which invariably begins with 5 pairs of fungiform papillae aligned along the central sulcus. Subsequently papillae appear more and more laterally, further anteriorly, and then further posteriorly.

Discussion

The two periods of rapid increase in fungiform papilla population are separated by a period of about two weeks duration during which the population size remains roughly constant. Farbman[7] and Mistretta[15] have both reported that in albino rats fungiform papillae are maturing during this period and neither reports seeing newly forming papillae after the first 2 or 3 days of extrauterine life.

Fish, Malone and Richter [8] failed to notice any changes in population size over the period from 8 hours to 466 days. However they took only 103 samples in that interval and the range of values they report is not very different from ours. The second population increase (Phase 2) has not to our knowledge been reported elsewhere. We do not yet know whether papillae, once formed, remain or whether some degenerate. There is a rapid turnover of cells in the adult taste bud. Beidler and Smallman [4] report that taste bud cells have an average lifetime of 250 ± 50 hours, but there is no indication that papillae have a similar fast turnover rate. If they do not then we have to conclude that Phase 2 mainly represents the maturation of a new set of papillae which began their development some 10 to 15 days after those of Phase 1.

These periods of increase in net papilla population coincide with changes in the manner of nutrient intake, the first occurring in a very brief period at birth when suckling is begun, the second taking place more gradually over the third and fourth weeks of postnatal life during the transition from suckling to self-feeding. The presence of this temporal relationship between development and behaviour prompted our next experiment. If the papilla population is changing during the transition to self-feeding, it is reasonable to suppose that there may be some interaction between feeding behaviour and papilla development.

EXPERIMENT 2: EARLY AND LATE WEANING

Methods

Normally, young rats begin about day 16 to sample very small quantities of solid foods, gradually increasing the amount consumed until solids constitute the whole diet and, over the same period, ingesting decreasing proportions of mother's milk. We decided to alter this pattern of weaning

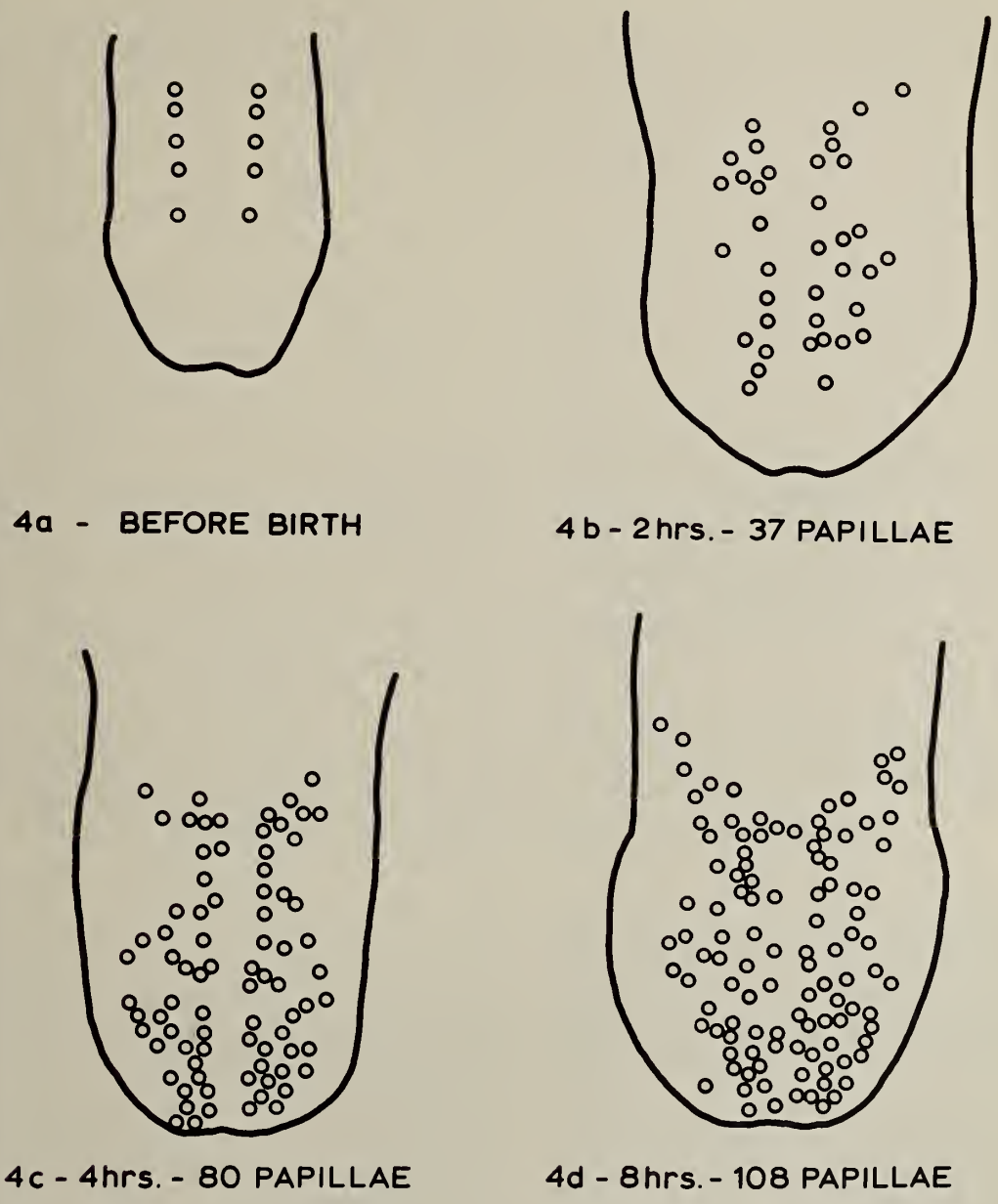


Figure 5–4. Maps of the distribution of non-staining papillae during Phase 1.
4a Prenatal and immediately postnatal distribution. Five pairs of papillae are ranged along the central sulcus, in mid-tongue.
4b Two hours postnatal. Increase is both lateral and anterior in direction. Tip of tongue is still unpopulated.
4c Four hours postnatal. Papillae are now in evidence at tip of tongue. Distribution is denser in mid-tongue.
4d Eight hours postnatal. Phase 1 is completed. The central sulcus remains free of visible papillae. This pattern of distribution and population size is found on all tongues up to the beginning of Phase 2.

by establishing three groups of four litters each as follows:

Early Weaning groups were separated permanently from their mothers on day 15, weaned first to a soft diet of Pablum and Esbilac (a commercial milk substitute), and then to standard laboratory rat chow.

Late Weaning groups were not allowed access to any solid foods, mother's faeces, or water for the duration of the experiment. Their mothers were fed away from the home cage in three 1-hour periods daily, the water bottle in the home cage was suspended above the reach of the pups, and the litters' total food intake consisted of mother's milk.

Control litters were allowed to wean normally with food and water available.

Samples from each condition were mapped and counted on days 17, 20, 24, and 28.

Results

Normally weaned controls have completed Phase 2 by day 24. As can be seen in Figure 5–5 those animals in the Early Weaning condition show an accelerated rate of increase in papilla numbers, completing Phase 2 by day 20. In the Late Weaning condition, the rate of increase in numbers is conversely affected, and the full complement of papillae has not been attained by day 28. There is no evidence that the final number in the Early Weaning group significantly exceeds that in the normals; the number in the Late Weaning group is still rising at the end of the experiment but shows no sign of failing to reach the normal level.

Discussion

By altering weaning conditions we have succeeded in changing the rate of increase but not the final number of papillae in this period of development, so that completion of Phase 2 in the two experimental conditions has been separated by more than 8 days. The explanation for these changes could be that ingestion of solid food stimulates the maturation of a second population of papillae. It could also be that mechanical or other effects associated with the suckling or mothering process retard the maturation of the second population. A third possibility is that a factor in maternal milk suppresses maturation. If either of the last two factors is operating its effect must diminish with age as Phase 2 does occur in the Late Weaning group although its onset is delayed and its rate slowed. The diminishing effect may be due to loss of susceptibility of the tongue epithelium to such factors or to the disappearance of the inhibiting agent from the milk. These two possibilities can be distinguished by cross fostering pups with mothers which gave birth either earlier or later in time than the birth date of the pups.

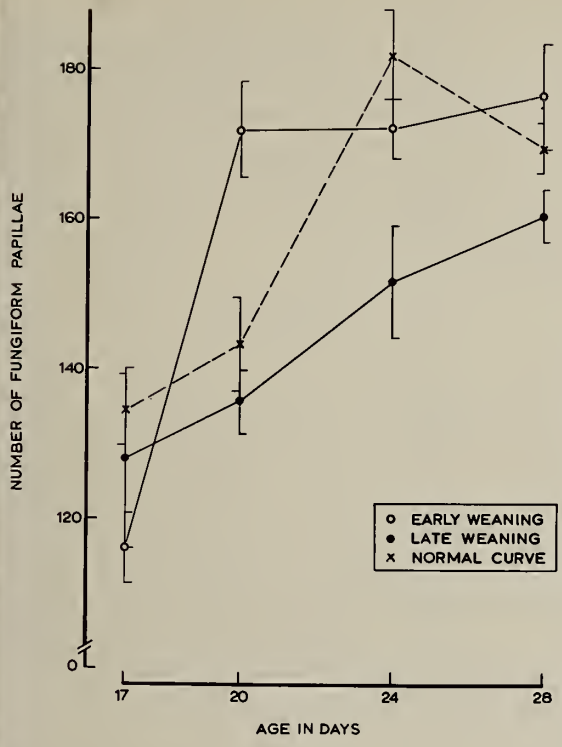


Figure 5–5. The effects of weaning conditions on Phase 2 of papilla population development.
Control group, weaned normally, complete Phase 2 around day 24.
Early Weaning group, weaned at day 15, show accelerated development, completing Phase 2 around day 20.
Late Weaning group, not weaned by day 28, show retarded development, Phase 2 not completed by day 28.
Weaning procedures affect the *rate* of development.

EXPERIMENT 3: EFFECTS OF CROSS FOSTERING LITTERS WITH MOTHERS OF DIFFERENT POST PARTUM AGES

Methods

Again three groups of litters were established as follows:

Late lactating mother (LLM) group in which at one day of age the pups were fostered to mothers which were six days post partum.

Early lactating mother group in which the pups were kept for 11 days with their biological mother and then fostered to a mother which was one day post partum. Part of this group was maintained by this mother until weaning (ELM 1) while another part was cross-fostered again on the 16th day of age to another mother which was 1 day post partum (ELM 2).

Control group reared in the usual manner with their biological mothers. Mothers in all experimental groups reared a mixed litter of 12 with pups

from each condition. No weaning constraints were imposed on any of the groups and lab chow and water were always available.

Samples from each condition were counted as before on days 17, 20 and 24.

Results

The results are plotted in Figure 5–6 and show that in pups raised by mothers of older post partum age Phase 2 was advanced while in those raised by mothers of earlier age it was retarded. The second cross fostering moves the maturation curve later still.

Discussion

These results give evidence that the maturation of papillae is suppressed either by some aspect of the suckling experience or by some component of maternal milk when the mother is in the early stages of lactation, and that this influence wanes with the post partum age of the mother. It is still possible that the important variable is weaning or experience with solid food. There is no evidence that weaning or self feeding occur earlier in animals nursed by late lactating mothers than in animals reared by their biological mothers. Yet these possibilities have by no means been eliminated. One further important possibility is that the effect on papilla maturation is part of a general effect on maturation exerted by undernutrition or deficiencies in maternal care in one or the other of the experimental conditions. Early lactating mothers may well provide inappropriate maternal care or milk of insufficiently

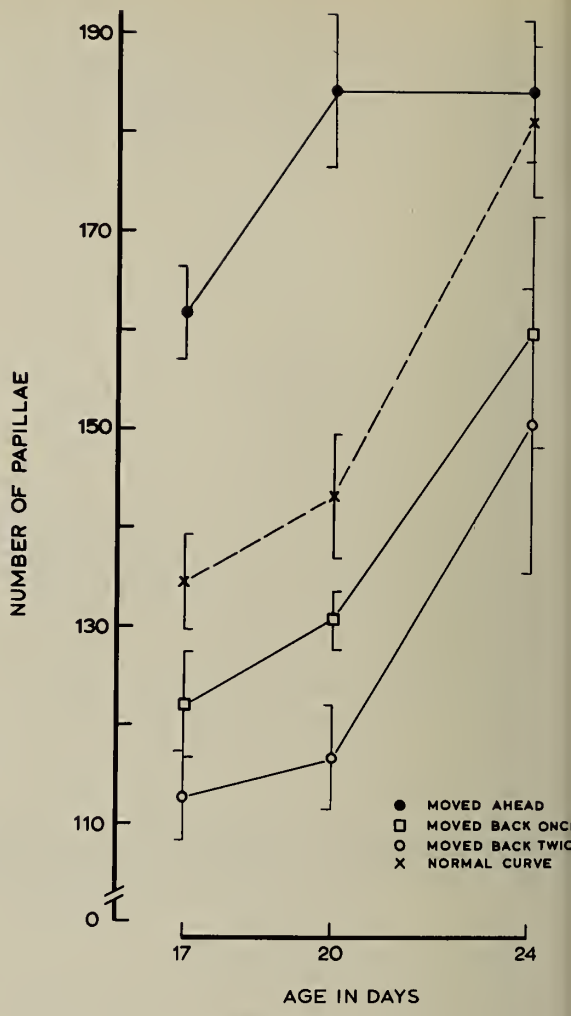


Figure 5–6. The effects of cross fostering to mothers of different postpartum ages on Phase 2.

Control group, reared by their own mothers, complete Phase 2 around day 24.

LLM group (moved ahead), reared by mothers of later postpartum age, complete Phase 2 around day 20.

ELM-1 group (moved back once), reared by mothers of earlier postpartum age, have not completed Phase 2 by day 24.

ELM-2 group (moved back twice), reared by two mothers, of earlier then again earlier postpartum ages, are further behind in Phase 2 development than ELM-1 group.

Fostering procedures affect development of Phase 2. The changes appear to be displacements in time, not in rate, as all curves are approximately parallel.

high quality or quantity to support the demands of older pups.

The last experiment in the series was an attempt to assess the effects on general pup development of an extensive cross fostering design with mismatching of pup and maternal post partum ages, and to distinguish the influence of age of milk from that of maternal care.

EXPERIMENT 4: CONTROL FOR GENERAL EFFECTS OF CROSS FOSTERING

Methods

Five groups of litters were established with the object of arranging that some animals were nursed by an early lactating mother (ELM) (1–6 days postpartum) throughout the study and others were nursed by a late lactating mother (LLM) (21 to 25 days postpartum) throughout.

To separate milk from maternal care the thelectomized biological mother (with nipples destroyed by cauterization) alternated with a normally lactating foster mother on a 3-6-3-3-6-3 hour schedule. Matching control groups were established in which the thelectomized foster mothers alternated with lactating biological mothers, and a final control group were reared by their biological mothers throughout.

Results

The effects of these manipulations on a number of measures of growth and maturation are shown in Figures 5–7,8 and 9. Regrettably a problem developed in the laboratory late in this experiment and it was not possible to complete the papilla counts. However, such data as we do have are consistent with the results of the previous studies, and the main thrust of the experiment, namely the effects of the design on general maturation, survives. In all cases, the experimental groups show developmental disadvantages compared with their appropriate controls and weight gain is slower. These control groups do not, however, differ from the normally reared third control group on any of the measures. Clearly therefore the cross fostering design, 12 hours per day food deprivation with thelectomized mothers, does not measurably disturb maturation. Maturation is quite severely disturbed, however, if the postpartum age of the lactating mother fails to match the age of the pups.

Discussion

The effects on papilla development of mismatching the postpartum age of the mother rat and her pups differ from the effects on other measures of somatic and neural development. Whereas the pups are disadvantaged in other respects by mismatches in either direction, Phase 2 of the development of the fungiform papilla population is specifically advanced when pups are reared by mothers in a later postpartum stage.

It is possible that the general retardation of development could be due to an inadequate supply of milk in both groups. High circulating prolactin levels are necessary for sustained milk production in rats [11] and prolac-

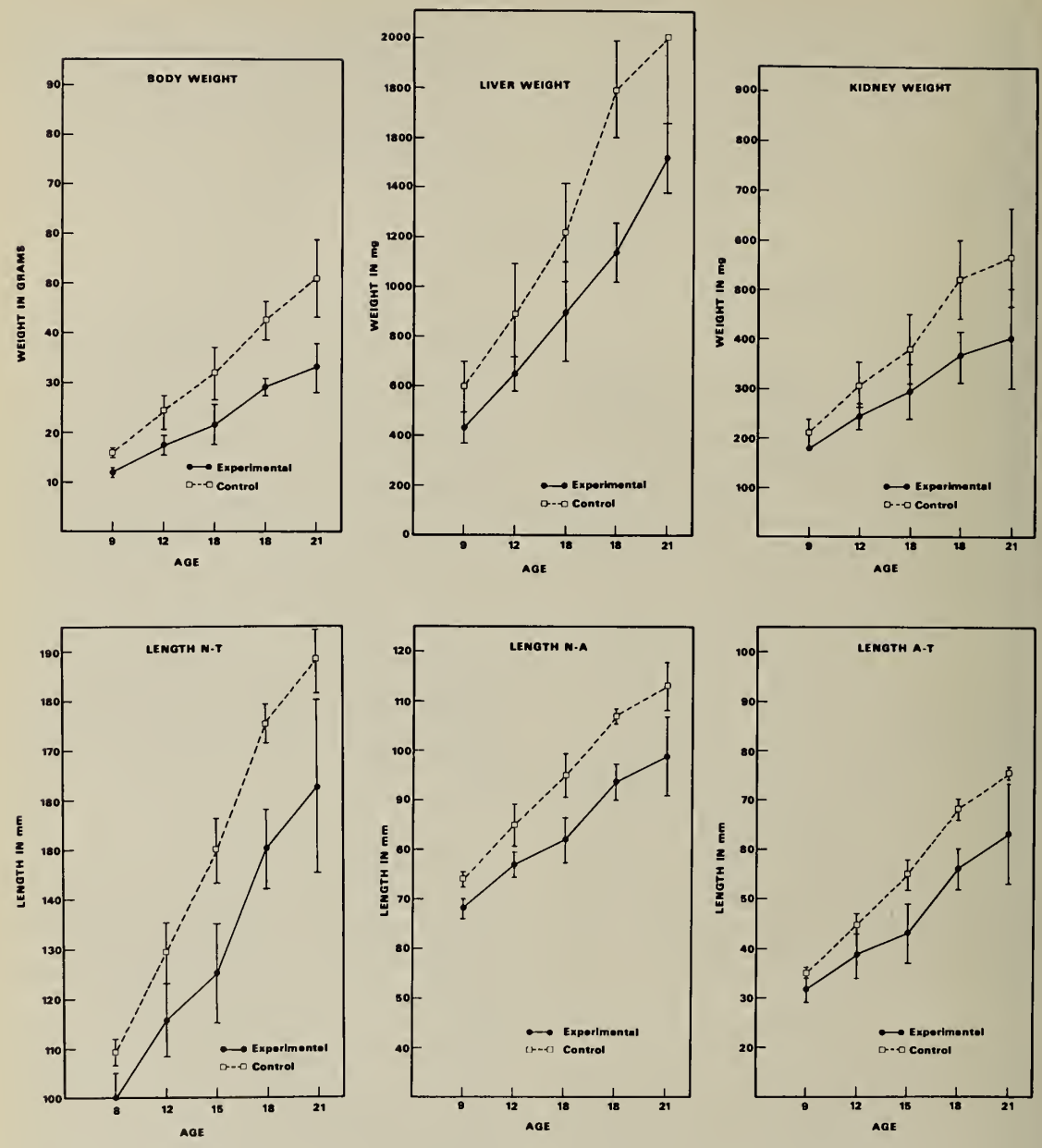


Figure 5–7. General somatic effects of cross fostering where pup age is grossly mismatched to mother's postpartum age. Experimental animals, whether reared by early lactating or late lactating mothers, lag behind controls in body weight, organ weight and skeletal growth. Skeletal length measures, nose to tail (N-T), nose to anus (N-A), and anus to tail (A-T), all correlate highly (0.95± 0.02) with body weights within each group, as do organ weights.

tin production in postpartum rats is, in turn, maintained by the stimulus of suckling young.[11,10,1] It seems possible that young rats do not provide a suckling stimulus to older mothers which is sufficiently intense to pro-

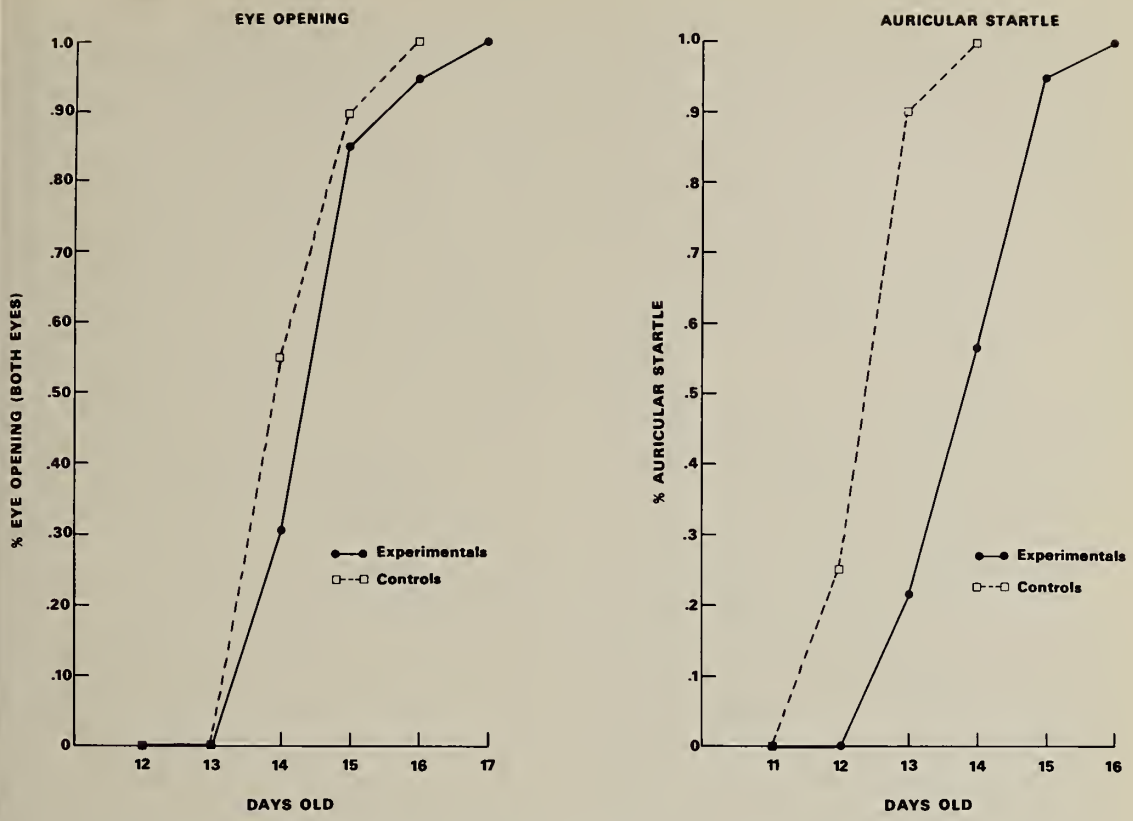


Figure 5–8. Measures of neural development.
Eye opening in animals mismatched to the mother's postpartum age occurs 24 hours later than in normally reared controls.
Auricular startle response is present in all normally reared controls by day 14, but not in all experimental animals until day 16.

voke secretion of prolactin at rates high enough to sustain adequate milk production. Moltz and Leon[16] have noticed a failure of the prolactin dependent maternal pheromone when lactating mother rats raise pups of different postnatal age. However this may not be the whole story. The maturationally retarded pups produced in this experiment seem to differ from runts suffering from undernutrition in large litters for example. In the retarded pups in our experiment there is a high correlation (0.97) between body weight and measures of skeletal growth. Unlike litter runts these animals appear to be perfectly proportioned but simply younger than their chronological age.

CONCLUSIONS

We have seen in Experiment 1 that a second period of growth of the fungiform papilla population occurs in hooded rats at the normal time of weaning and sampling of solid food. From Experiment 2 we see that this second phase can be altered by changing the time of weaning but not in-

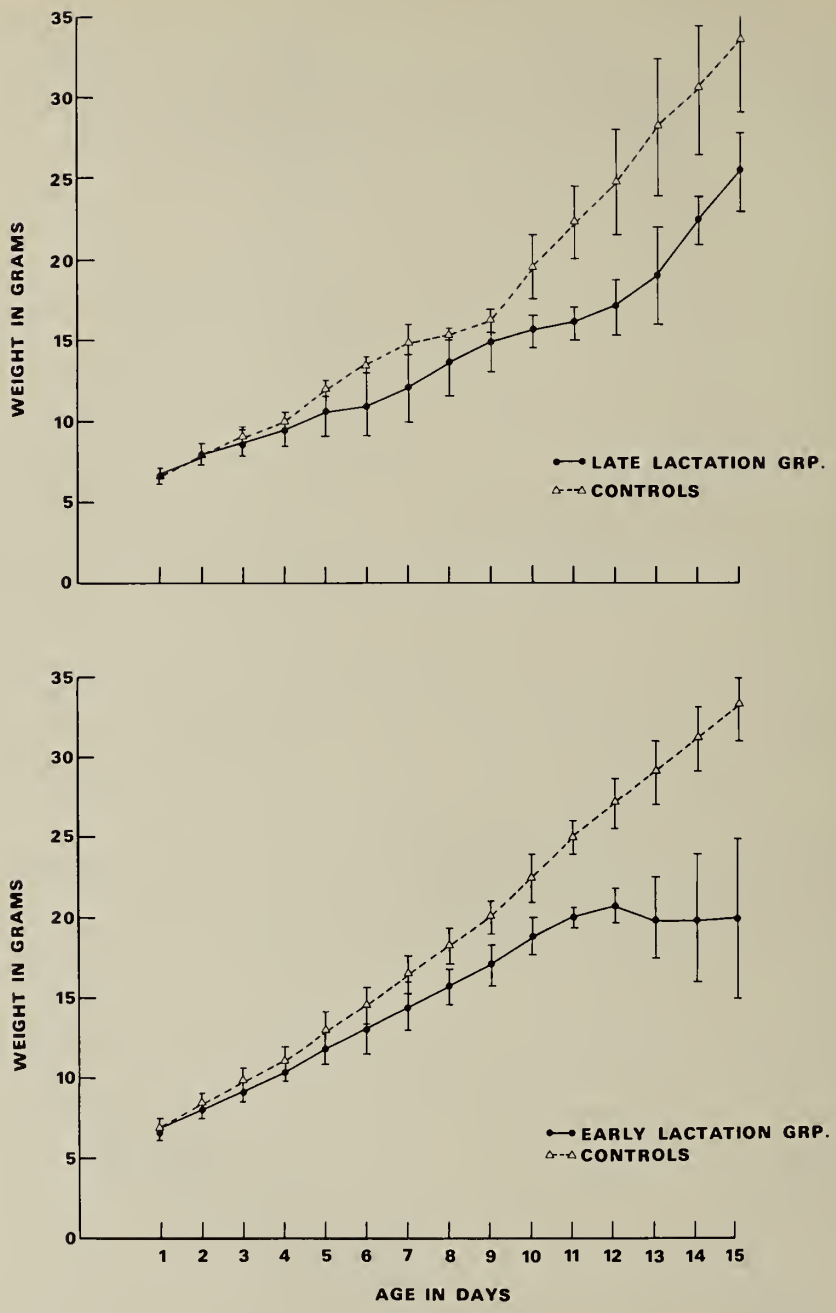


Figure 5–9. Comparisons of mean litter weights for each experimental group with its matching control group. Differences between experimental and control groups in both the late lactating mother condition and in the early lactating mother condition are evident by day 6, and are statistically significant by day 9. There are no significant differences between LLM experimental and ELM experimental groups. Nor are there significant differences between LLM control and ELM control groups. Both of these control groups show weight gains essentially like those of litters reared normally by their own mothers.

definitely forestalled. Experiment 3 demonstrates that the controlling factor is not associated with the first experiences with solid food but with either the suckling-mothering experience or with maternal milk. Finally the results of Experiment 4 allow us to conclude that the changes in the second phase are not due to effects of cross fostering on maturation in general but to a specific inhibitory effect on papilla development of a component either of the suckling-mothering experience or of the milk. We are unable, at present, to distinguish between these two possibilities although the latter seems, at the moment, more likely. We are preparing to do a series of artificial feeding experiments which we hope will make this important distinction clear.

The results raise interesting questions in a number of areas. Firstly it is interesting to have found an environmental influence upon the development of epithelial tissue and in turn, since fungiform papillae are apparently the precursors of taste buds, upon the development of the taste system itself. We have, of course, no evidence to indicate what the nature of this influence may be, whether mechanical or chemical, nor do we know whether the fungiform papillae which form during the second phase differ from those which appear earlier either morphologically or functionally. All of these questions seem to suggest potentially fruitful areas to pursue.

Perhaps equally interesting are the questions of the interaction between morphological development and behaviour raised by our results. For example how does the appearance of the second phase of population growth affect the animal's ability to form taste preferences and aversions? Does the second phase represent the development of a set of receptors with different properties—properties which perhaps are influenced by the animal's early experiences with food? Or again how might the properties of a second population of receptors influence the development of feeding behaviour in the animal?

REFERENCES

1. Amenomori, Y., Chen, C.L., and Meites, J.: Serum prolactin levels in rats during different reproductive states. *Endocrinology,* 86: 506–510, 1970.
2. Baradi, A.F., and Bourne, G.H.: Gustatory and olfactory epithelia. *International Review of Cytology,* 2:289–330 1953.
3. Beidler, L.M.: Dynamics of taste cells. In Zotterman, Y. (Ed.) *Olfaction and Taste,* Oxford, Pergamon, 1963, pp. 133–145.
4. Beidler, L.M., and Smallman, R.L.: Renewal of cells within taste buds. *J. Cell. Biol.,* 27:263–272, 1965.
5. Beidler, L.M.: Innervation of rat fungiform papilla. In Pfaffmann, C. (Ed.), *Olfaction and Taste III,* New York, Rockefeller, 1969, pp. 352–369.
6. von Ebner, V.: Handbuch der Gewebelehre des Menschen. Band 3, Koelliker, 1902.
7. Farbman, A.I.: Electron microscope study of the developing taste bud in rat fungiform papilla. *Dev. Biol.,* 11:110–135, 1965.
8. Fish, H.S., Malone, P.D., and Richter, C.P.: The anatomy of the tongue of the domestic Norway rat. I. The skin of the tongue, the various papillae; their number and distribution. *Anat. Rec.,* 89:429–440, 1944.

9. Galef, B.G., and Henderson, P.W.: Mother's milk: a determinant of feeding preferences of weaning rat pups. *J. Comp. Physiol. Psychol.* 78: 213–219, 1972.
10. Grosvenor, C.E., Maiweg, H., and Mena, F.: A study of factors involved in the development of the exteroceptive release of prolactin in the lactating rat. *Hormones and Behavior,* 1:111–120, 1970.
11. Grosvenor, C.E., Mena, F., Dhariwal, A.P.S., and McCann, S.M.: Reduction of milk secretion by prolactin-inhibiting factor: Further evidence that exteroceptive stimuli can release pituitary prolactin in rats. *Endocrinology,* 81:1021–1028, 1965.
12. Guth, L.: The effects of glossopharygeal transection on the circumvallate papilla of the rat. *Anat. Record.,* 128:715–732, 1957.
13. Guth, L.: Taste buds on the cat's circumvallate papilla after reinnervation by glossopharyngeal, vagus and hypoglossal nerves. *Anat. Record.,* 130:25–38, 1958.
14. May, R.M.: The relation of nerves to degenerating and regenerating taste buds. *J. Exp. Zool.,* 42:371–410, 1925.
15. Mistretta, C.M.: Topographical and histological study of the developing rat tongue, palate and taste buds. In Bosma, J.F. (Ed.) *Third Symposium on Oral Sensation and Perception,* Springfield, Thomas, 1972, pp. 163–187.
16. Moltz, H., and Leon, M.: Stimulus control of the maternal pheromone in the lactating rat. *Physiol. and Behaviour,* 10:69–71, 1973.
17. Oakley, B.: Altered taste responses from cross-regenerated taste nerves in the rat. In Hayashi, T. (Ed.), *Olfaction and Taste II,* Oxford, Pergamon, 1967, pp. 535–547.
18. Oakley, B.: On the specification of taste neurones in the rat tongue. *Brain Research,* 75:85–96, 1974.
19. Olmsted, J.M.D.: The nerve as a formative influence in the development of taste buds. *J. Comp. Neurol.,* 31:465–468, 1920.
20. Olmsted, J.M.D.: The results of cutting the seventh cranial nerve in Ameriurus Nebulosis. *J. Exp. Zool.,* 31:369–401, 1920.
21. Olmsted, J.M.D.: Effects of cutting the lingual nerve of the dog. *J. Comp. Neurol.,* 33:149–154, 1921.
22. Olmsted, J.M.D.: Taste fibres and the chorda tympani. *J. Comp. Neurol.,* 34:337–341, 1922.
23. Torrey, T.W.: The relation of taste buds to their nerve fibres. *J. Comp. Neurol.,* 59:203–220, 1934.
24. Torrey, T.W.: The relation of nerves to degenerating taste buds. *J. Comp. Neurol.,* 64:325–336, 1936.
25. Torrey, T.W.: The influence of nerve fibers upon taste buds during embryonic development. *Proc. Natl. Acad. Sci.,* 26:627–634, 1940.
26. von Vintschgau, M.: Beobachtungen Uber die Veranderungen der Schmeckbecher nach Durchniedung des N. Glossopharyngeus. *Arch. Ges. Physiol.,* 23:1–13, 1880.
27. von Vintschgau, M., und Honigschmied, J.: Nervus glossopharyngeus und schmeckbecher. *Arch. Ges. Physiol.,* 14:443–448, 1877.
28. Whiteside, B.: Nerve overlap in the gustatory apparatus of the rat. *J. Comp. Neurol.,* 44:363–377, 1927.
29. Zalewski, A.A.: Changes in phosphatase enzymes following denervation of the vallate papilla of the rat. *Exp. Neurol.,* 22:40–51, 1968.
30. Zalewski, A.A.: Combined effects of testosterone and motor, sensory or gustatory nerve regeneration of taste buds. *Exp. Neurol.,* 24:285–297, 1969.
31. Zalewski, A.A.: Neurotrophic-humoral interaction in the regulation of taste buds in the rat's vallate papilla. *J. Neurobiol.,* 1:123–132, 1969.
32. Zalewski, A.A.: Regeneration of taste buds after reinnervation by peripheral or central sensory fibres of vagal ganglia. *Exp. Neurol.,* 25:429–437, 1969.
33. Zalewski, A.A.: Regeneration of taste buds after transplantation of tongue and ganglia grafts to the anterior chamber of the eye. *Exp. Neurol.,* 35:519–528, 1972.
34. Zalewski, A.A.: Regeneration of taste buds in tongue grafts. *Exp. Neurol.,* 40:161–169, 1973.

DISCUSSION

MISTRETTA: I am concerned about the accuracy of your method for identifying and counting papillae on the developing rat tongue. Are you certain that you can recognize papillae when they first appear? Farbman [1] and Mistretta [2] have both reported the first appearance of fungiform papillae on the tongue of the *fetal* rat between 15–16 days of gestation. These reports were based on transmission electron microscopy and scanning electron microscopy, respectively. Torrey [3] reported the first appearance of the circumvallate papilla on the fetal rat tongue at 15 days of gestation. Both Farbman and Mistretta noted that the distribution of fungiform papillae on the fetal tongue corresponds to that on the adult tongue. The number of taste pores (an index of number of mature taste buds), however, increases gradually over the first 12 days after birth.[2] Perhaps your method is not sensitive enough to recognize papillae on the fetal tongue.

HENDERSON: The primary aim of this work was to determine if papilla populations in young animals were different to those in adults. We are aware of reports in the literature that taste buds are not present during the first week of postnatal life, but the assumption made here was that the number of designated sites might be established, not that they were occupied. Determination of taste bud populations must, of course, be by histological procedures, and we are planning a systematic examination of both Phase 1 and Phase 2 papillae by such methods.

MISTRETTA: And you have never seen them on a fetal tongue?

HENDERSON: No, only the first five pairs which appear during labour. There may be partially formed papillae not yet evident by surface examination. However, magnification of 312 should be adequate for detecting structures of this size, and the regularity of development coupled with an extremely small variability in these counts would seem to bear this out. Further, the rats used in these studies are the hooded form, not albino. We have begun comparison studies with rats of an albino strain and the developmental pattern from day 1 to day 25 in these animals is extremely variable. This variability may well be reflected in the prenatal period as well.

---

[1] Farbman, A.I. Electron microscope study of the developing taste bud in rat fungiform papilla. *Dev. Biol.* 11:110–135, 1965.

[2] Mistretta, C.M. Topographical and histological study of the developing rat tongue, palate and taste buds. In Bosma, J.F. (Ed.) *Third Symposium on Oral Sensation and Perception*, Springfield, Thomas, 1972, pp. 163–187.

[3] Torrey, T.W. The influence of nerve fibers upon taste buds during embryonic development. *Proc. Nat'l. Acad. Sci.* 26:627–634, 1940.

Chapter 6

THE NEONATAL PIG: A BIOLOGICAL MODEL FOR THE DEVELOPMENT OF TASTE PREFERENCES AND CONTROLS OF INGESTIVE BEHAVIOR

KATHERINE A. HOUPT AND T. R. HOUPT

Nearly all of the work presented in this volume has been done on rats, other rodents, or humans. Although others have previously made similar pleas for the use of various species, we will again raise the question of selecting an appropriate biological model. We will argue that the pig is an exceptionally good model for the human in the study of functional phenomena associated with the alimentary canal (see also[2, 3, 8])

SIMILARITIES AND DIFFERENCES

The most obvious similarity of pig and man is body size. The laboratory rat is two orders of magnitude different from the human in this respect. The ordinary pig as it matures does exceed the weight of the average person, but still maximum body weight will only be about 2 or 2½ times average human weight—and there are a few people at that elevated level. So called miniature pigs, although not really so small, are, when mature, even more comparable to man. This question of scale has general metabolic consequences. One is that the smaller animal—such as the rat—generally matures in a few weeks, while the large ones—for example, man—may not do so for many years. Yet the pig matures within the year, combining similar body size to the human with the rapid growth necessary for convenient completion of many experimental procedures.

The diet of man and pig is also rather similar, nearly identical for garbage-fed pigs. If one surveys the common laboratory and domestic mammals, the pig is really the only naturally omnivorous mammal among our close companions. The rat has omnivorous habits—presumably in part the result of his close association with us and our food stores—but the alimentary canal, most strikingly the ridged herbivore-type molars, is not that of a typical omnivore.[1] The dog likewise has become an omnivore because of man's influence. However,

Acknowledgment: K.A. Houpt holds National Institutes of Health Special Fellowship 1-F03-AM-55321-01.

the sectorial teeth and simple carnivorous gastrointestinal tract belie its domestic omnivorous habits.

A brief look at the dentition of the pig may be of interest in this respect.[24] Of the teeth the molars are most similar to those of man, being of the bunodont form with separate cusps adapted for a triturating action on ingested foodstuffs rather than with cusps fused to form the ridged pattern of typical herbivores. (Figure 6–1) There are, of course, differences from the human arrangement. (Figure 6–2) The slanting incisors are almost horizontal in the lower jaw which is used by the pig as a sort of scoop in prehension of food. The canines of the boar (as in Fig. 6–1) are of the rootless type which grow continuously and have a large pulp cavity. The molars are in nearly parallel rows, unlike the nearly semicircular human dental arcade. The diastemata are another obvious difference. The dental formula is of the primitive mammalian form $I\ \frac{3}{3}\ C\ \frac{1}{1}\ P\ \frac{4}{4}\ M\frac{3}{3}$.

Figure 6–1. Skull of mature boar with bone removed to show embedded parts of the teeth. I, incisors; C, canines; P, premolars; M, molars. (From Sisson, S., and Grossman, J. D. The Anatomy of the Domestic Animals. 4th. Edition. Philadelphia, W.B. Saunders Co., 1953.)

Figure 6–2. Lower jaw of sow. Note extensively worn premolars.

It occurred to us that with regard to dentition perhaps the pig is a better model for the higher non-human primates rather than for man. In some respects the dental arrangement of the higher non-human primates differs significantly from that of man. In Figure 6–3 is shown the lower jaw of a baboon which can be compared to that of the pig in Figure 6–2. Note the parallel arrangement of cheek teeth, the slight slant of the incisors, the extra complexity of the last molar, and, of course, the large canine teeth. In these respects pig and baboon are similar to one another, but both are somewhat different from man. Hence the suggestion that pigs might be good substitutes for the higher non-human primates, which in turn are often presumed to be the ideal models for man.

As to the occurrence of dental caries, there is at least one report to the effect that caries could not be induced in pigs.[11] Our pathologist (Dr. John King) reports that although he

Figure 6–3. Lower jaw of young but mature male baboon.

and his associates routinely examine the teeth of all pigs on which postmorteum examination is made, he can not recall ever seeing any caries even in aged pigs.*

The remainder of the alimentary canal of the pig is also surprisingly similar to that of the human (Figure 6–4). The stomach, small intestine and large intestine are similar in general form to those of the human. The most notable differences are the slightly larger cecum and the longer large intestine of the pig. The complex spiral arrangement of the pig's intestine in the peritoneal cavity is also quite different.

Finally, we see similarity between people and pigs with respect to their regulation of the glucose concentration of extracellular fluid. This is particularly so in the newborn. The most striking example of this similarity is the proneness of newborn pigs and infants to develop a hypoglycemic syndrone. Hypoglycemia is common among fasted neonate pigs,[22] but generally is seen only in low birth weight infants where about 25% of the infants have abnormally low plasma glucose concentration.[23,4] The normal neonate pig resembles the low birth weight human infant in that it lacks fat and glycogen stores.[19] A failure of food intake under these circumstances can result quickly in a serious hypoglycemia. We are currently investigating the possible glucostatic controls of food intake in the neonate pig with an eye to the implications for the human infant and the role of sweet taste preference. We will report on this work in the second part of our paper.

*There is a clinical report of carious lesions in a 1½ year old boar which had been fed a 50% sucrose diet *ad libitum* (Andrew, A.H. *Vet. Rec.* 93:257, 1973.)

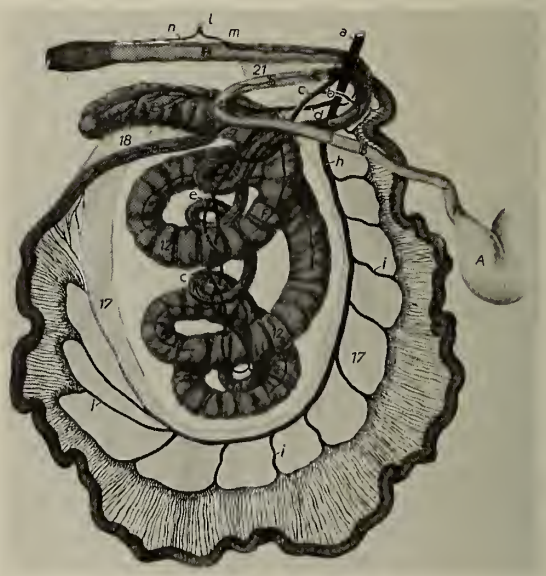


Figure 6–4. Intestinal tract of the pig. A, pyloric part of stomach; B (1–6), duodenum; C, jejunum; D, ileum; E, cecum; F, G, H, colon; J, rectum; a, cranial mesenteric artery and (b-k) branches; 1, caudal mesenteric artery and (m-n) branches. (From Nickel, R., Schummer, A., Seiferle, E. and Sack, W.O. The Viscera of the Domestic Animals. Berlin, Verlag Paul Parey, 1973.)

PIGS IN THE LABORATORY

The use of pigs in the laboratory does, of course, present some practical problems. Pigs are not the docile automata which laboratory rats often seem to be. For example, the pig will protest vigorously almost any procedure however innocuous. This largely vocal protest may call for some soundproofing of the laboratory. We have worked mostly with newborn or young pigs and housing them has been simple. Either dog or rabbit cages can be used with feeders more suitable for the pig's snout attached to the inside of the cage. For unweaned pigs we have supplied milk by bottle. Similac (Abbott) or SPFlac (Borden), an artificial sow's milk, are suitable. We have found that a simple rack of our own design attached to the cage side quite satisfactorily holds several infant bottles (Figure 6–5). The newborn pig cannot maintain its body temperature, and unless kept in a warm environment its body temperature will fall since the relatively large surface area of its body lacks insulation of either fur or subcutaneous fat.[20] For the first few days we have used either chick brooders or infrared heat lamps for this purpose. Later, simply keeping the room temperature at about 80°F suffices.

Figure 6–5. Piglet suckling from bottles held in rack.

Gastrointestinal disease can be an important problem when neonate pigs are kept under laboratory conditions. Newborn piglets are normally protected from enteric disease by immunoglobulins present in the sow's milk. It has been reported that the immunoglobulins present in colostrum which are absorbed by the neonate during the first 24 hours of life are most effective against systemic infection; while the antibodies present in milk during the following days are predominantly effective against enteric microorganisms, absorption of the antibodies being neither necessary nor desirable. The piglet is not able to form adequate amounts of immunoglobulin of its own until about 14 days of age.[25] We have had occasional cases of diarrhea develop in our newborn piglets housed in cages, and we suspect that it is due to enteric colibacillosis which develops because the normal source of enteric immunoglobulins are no longer being supplied to the piglet from the sow. Under these circumstances enteric bacteria normally present in the digestive tract may have pathologic effects. So far we have been reasonably successful in combating this problem by strict sterilization of feeding equipment and general cleanliness. Other methods

which likely would be more effective are also rather heroic: milking sows and giving the piglets the sow's milk by bottle. Parenthetically, one can add that it seems that the neonate pig is also a good model for infantile diarrhea (see also Ref. 14).

A rather irksome difference between pigs and other mammals of comparable size is the scarcity of superficial blood vessels for drawing blood or injecting solutions. We find that in order to place a plastic catheter into the jugular vein a minor surgical operation is required. Under general anesthesia the skin is incised and the rather small external jugular vein is exposed by blunt dissection through several millimeters of subcutaneous tissue. We lead the polyethylene catheter subcutaneously to an exit on the dorsal aspect of the neck. The catheter is filled with a sterile heparin-saline solution and sealed with a ligature. Daily rinsing with heparin-saline is generally adequate to keep the catheter clear. However, we have had some difficulty with blockage of the catheter, apparently by a tissue flap growing over the tip. When this happens, withdrawal of blood becomes tedious or impossible, but solutions can still be injected. The extremely rapid growth of piglets presents an unusual problem: the pig may outgrow the catheter and within a few weeks the tip slips out of the vein.

Kare [12] has stated that the localization of taste buds to the mouth and pharynx is common to all air breathing vertebrates and that the taste receptors are similar in many domestic animals—rat, dog, pig and calf. However, it is clear that the taste sensory patterns perceived centrally by animals of the various species and even between individuals of the same species differ. Presumably these sensory patterns are interrelated with, and subserve, the dietary habits and digestive physiology of each species.

TASTE PREFERENCES IN THE NEONATE PIG

Before considering development of sweet taste preference in neonatal pigs, we must first ascertain that adult pigs do prefer sweet solutions. Fortunately, pigs have been well stuided in this respect. Kare, Pond and Campbell [13] were the first to show that pigs given a choice between sucrose solution and water showed a strong preference for sucrose solutions of 1%(0.03 M) and higher concentrations. They also measured preference for glucose, lactose and saccharin, but individual variation among pigs tended to mask any differential preferences. The work of Kare, Pond and Campbell was carried further by Kennedy and Baldwin.[15] They plotted preference curves for glucose, sucrose, sodium cyclamate and saccharin based upon both 12 hour and 1 hour preference tests. Sodium cyclamate was not preferred at any molarity. Peak preference was about 0.1 M for the two sugars and for saccharin. At higher concentrations a definite aversion for saccharin developed. Kennedy and Baldwin[15] also showed that pigs can learn to perform an operant response in order to obtain sucrose solutions. The pigs' motivation to respond on progressively higher ratio schedules was related to the concentration of the sucrose

reinforcers. The more mature pig seems strongly to prefer sweet solutions and is willing to work for them. In these respects, as in many others, pigs are like people.

We wished to determine whether the neonatal pig like the human infant [5] shows a preference for sweet solutions. Piglets were removed from the sow at 48 hours of age after they had had ample time to ingest colostrum. The were placed in the specially modified cages and maintained on a commercially available artificial sow's milk (SPF1ac, Borden). Before each taste test the pigs were fasted for 3 hours. They were then given a two bottle preference test. One bottle held distilled water and the other the sugar solution. The positions of the bottles were reversed at each test to control for position preferences. The bottles were weighed at the beginning and end of the one hour test period to determine the amount drunk by the pigs. The results are shown in Figure 6–6. No preference was shown for 0.1 M glucose in comparison to water, but marked preference was seen for 0.3 M and 0.5 M glucose and for 0.5 M sucrose. Mean intake of water was less than 20 g/hr in

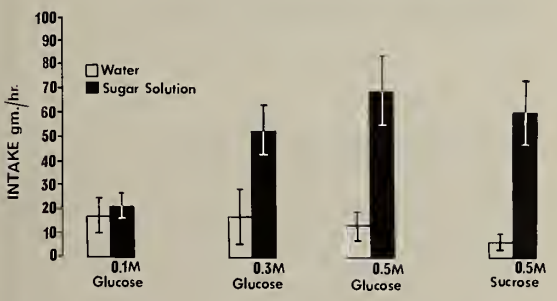


Figure 6–6. Intake of water and various sugar solutions by suckling pigs in a two bottle preference test.The height of each column represents the mean ± SEM. There are 10 observations for each condition.

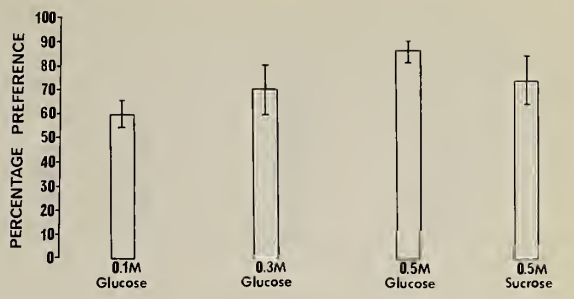


Figure 6–7. The percentage preference for various sugar solutions by suckling pigs. There are 10 observations for each condition.

each case. Mean intake ± SEM of 0.1 M glucose was 20.9±4.6 g/hr; that of 0.3 M glucose, 52.0±12.9; that of 0.5 M glucose, 68.3±15.1; and that of 0.5 M sucrose, 59.9±13.6. Figure 6–7 shows the percentage preferences for the four sugar solutions tested calculated as: (sugar solution consumed/total fluid consumed) X 100. The percentage preference for 0.1 M glucose was only 59%, but that for 0.3 M glucose was 71% and that for 0.5 M glucose was 86%. Preference for 0.5 M sucrose was 73%.

In addition to measuring intake, we also observed the animals during the taste preference tests whenever possible. We used a stopwatch to record the duration of sucking bouts and tallied contacts of the pigs with the bottles. Figure 6–8 shows the contacts (sucks or licks) that were made with the bottle containing the sugar solution. The pigs made 9.7±2 contacts (mean ± SEM per hour) with 0.1 M glucose versus 6.3±1 contacts with water; 16.6±4 contacts with the 0.3 M glucose bottle versus 6.0±3 contacts with water; and 12.8±2 contacts with the 0.5 M glucose versus 4.2±1 contacts with water. Finally, they made 8.8±2 contacts with the 0.5 M sucrose bottle versus 4.4±1 contacts with wa-

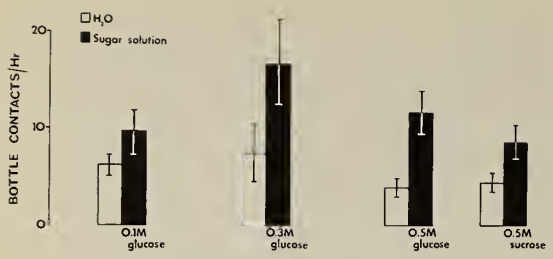


Figure 6–8. Bottle contacts per hour by suckling pigs during two bottle preference tests. Touching the nipple with the snout and/or sucking on the nipple constitutes a contact. The number of observations per condition equals 11 for 0.1 M glucose, 5 for 0.3 M glucose, 6 for 0.5 M glucose and 9 for 0.5 M sucrose.

ter. Figure 6–9 shows the duration of sucking on each type of fluid during the one hour test period. This is the individual record for one pig. Although this pig spent less than one minute sucking on the water bottle in each two bottle choice situation, it spent 35 minutes sucking on the bottle containing 0.5 M glucose.

The amount consumed, the percentage preference, the number of mouth contacts with the bottle nipples, and the duration of sucking all indicate that that neonatal pig has already developed a marked sweet preference. It is interesting to note that although the suckling pig has a distinct preference for glucose, this preference is not shown until the concentration is 0.3 M glucose or higher, whereas the more mature pigs studied by Kennedy and Baldwin [15] showed a strong preference at 0.05 M glucose. Apparently, the neonatal pig is not as sensitive to glucose as is the adult pig.

Seeking some point of contact with the study of sweet preference in children, we offered to a piglet a sweet solution which more closely resembles those in the environment of a child. Following a three hour fast, the piglet, drinking from open dishes, was given a two choice perference test between water and a commercially available orange-flavored soft drink which is approximately 0.3 M sucrose. In two trials it consumed an average of 112 g orange drink versus 30 g of water. It would appear that pigs, like children, prefer sweetened beverages.

The piglet may display some nutritional wisdom. In a two bottle, one hour preference test between 0.5 M glucose and artificial sow's milk (SPFlac, Borden), piglets that have been fasted for three hours, preferred the milk. They drank an average of 110 g milk and 54 g of the sugar solution.

It seems clear that the suckling pig possesses a marked sweet taste preference, but is the preference an innate characteristic present from birth or is it learned post-natally? Foster [7] attempted to answer this question. He used 6 newly born pigs which had not yet nursed, placed each pig in an individual cage, and offered each a choice

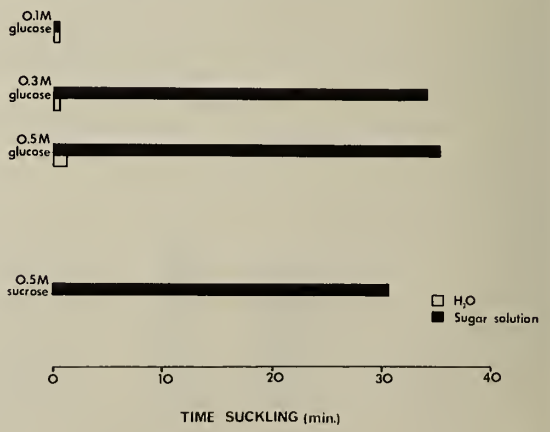


Figure 6–9. The time spent sucking by one piglet during each of 4 two bottle preference tests. When given a choice between water and 0.5 M sucrose, the piglet sucked only on the sucrose-containing bottle.

between water and 1.3% saccharin for 24 hours. He found that 72% of the total fluid consumed by the piglets was from the saccharin container and only 28%from the water. Foster's failure to report total volume of fluid intake or environmental temperature, which should be high for normal behavior to occur, severly limits the usefulness of his data.

GLUCOPRIVATION AND FOOD INTAKE

Our interest in the development of the controls of food intake in the neonatal pig spans not only taste preferences but also the elicitation of eating in response to reduced levels of metabolizable energy. Mature animals of many species increase their food intake in response to a lack of metabolizable glucose.[17,6,10] One method of producing a deficit of glucose to the brain, i.e., glucoprivation, is by administration of exogenous insulin. This is the method we chose to use in pigs.

Eating in response to glucoprivation has not been previously demonstrated in pigs, although Pekas et al.[21] reported negative results when insulin and tolbutamide were used in an attempt to stimulate eating. We first had to find a dose of insulin sufficient to produce a profound hypoglycemia, but not so high as to incapacitate the animal. We used pigs with indwelling jugular catheters as described above. The plasma glucose response to 0.5 and 1.0 units/kg body weight of regular insulin was determined on blood samples taken just before insulin injection and hourly for 6 hours after injection. Figure 6–10 shows the results. Both 1.0 and 0.5 units/kg insulin caused plasma glucose to fall from 100 mg/100 ml plasma to less than 40 mg/100 ml, and plasma glucose remained low for at least 3 hours following the injection. 1 unit/kg insulin is apparently more than adequate to produce glucoprivation. We found that 2.0 units/kg was too high a dose because it produced convulsions and in one case death. It is interesting to compare the sensitivity of the pig to insulin to that of the rat. The rat can tolerate a dose of 8 units/rat as long as food is available. Such a dose (greater than 16 units/kg) would surely kill a pig. Pigs more closely resemble dogs and people in their sensitivity to insulin.

In the experiments on the feeding responses to insulin, 5 female Yorkshire pigs, 4–8 weeks of age, were tested. They were housed individually in dog metabolism cages and fed a commercial pelleted pig food ad libitum. All spillage was collected and measured. The pigs were injected daily intraperitoneally with either insulin or an equivalent volume of 0.9% NaCl as a control. Food intake was measured every 2 hours for 6 hours following the injection. Twenty-four

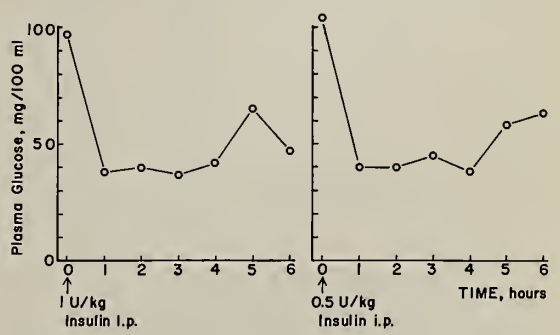


Figure 6–10. The effect of insulin on plasma glucose of a weanling pig.

hour intake was also measured. Figure 6–11 gives the results. Baseline intake was 143±12 g/6 hr. (mean±SEM). Intake was increased by 0.75 units/kg regular insulin, but the increase was not statistically significant. 1 unit/kg insulin significantly increased intake to 205±21g/6hr ($t=2.6$; $p<0.02$). Insulin apparently stimulates an increase in short-term food intake in weanling pigs. Figure 6–12 shows the daily 6 hr intake of a 6 week old male pig kept in a different laboratory. Baseline (saline injection) intake tended to increase as the animal rapidly grew from 16 to 25 kg body weight in 2 weeks. Nevertheless, whenever exogenous insulin was administered, intake increased. Mean intake (±SEM) following 1.0 unit/kg insulin was 436±45 g and that following 0.9% NaCl was 274±31 g ($t=2.8$; $p<0.025$). Insulin did not affect 24 hour intake in any of the pigs tested, and there appear to be no sex differences in the response to insulin. Relatively mature pigs as so many other mammals studied display glucoprivic eating.

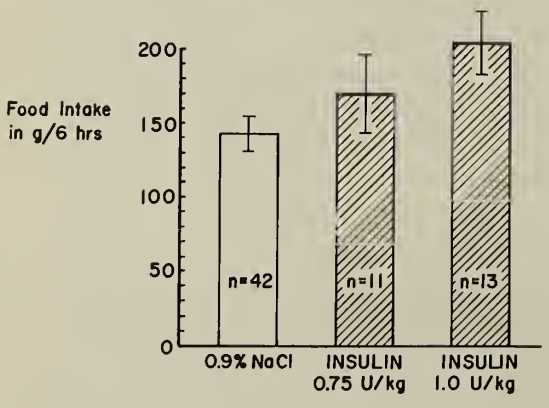


Figure 6–11. The effect of insulin on short term food intake of weanling pigs. N is the number of observations. 0.75, 1.0 units/kg of regular insulin, or equivalent volume of 0.9% NaCl were injected intraperitoneally.

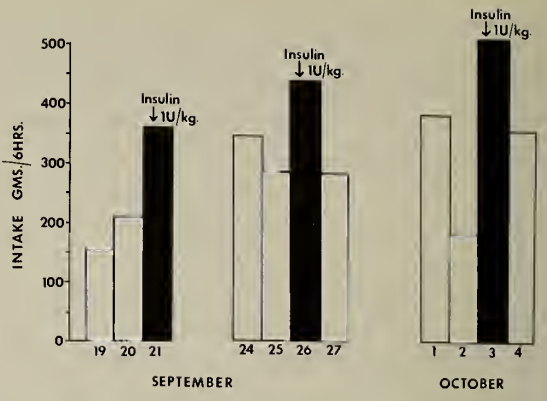


Figure 6–12. The individual record of pig L showing 6 hour intake following 0.9% NaCl injection (open columns) or 1.0 unit/kg regular insulin (solid columns).

We were particularly interested in testing pigs within the first two or three days of life because this is the time when they are most susceptible to neonatal hypoglycemia from a variety of pathological causes. We wished to produce the hypoglycemic syndrome with exogenous insulin and to measure the ingestive response. Because we used piglets which were nursing sows, intake could not be measured directly. Instead we used short-term, six hour, weight gain as a measure of food intake. Each pig was weighed individually on a triple beam balance, injected with 1.0 units/kg or 2.0 unit/kg regular insulin or with an equivalent volume of 0.9% NaCl, and returned to the sow. At the end of 6 hours the piglets were reweighed. Twenty-four hour weight gain was also measured, but no significant differences were found between the groups. Figure 6–13 illustrates the effect of insulin on weight gain (i.e., milk intake) of neonatal pigs. Saline injected pigs gained 24±5 g/6 hr, while littermates injected with 1.0 unit/kg insulin gained

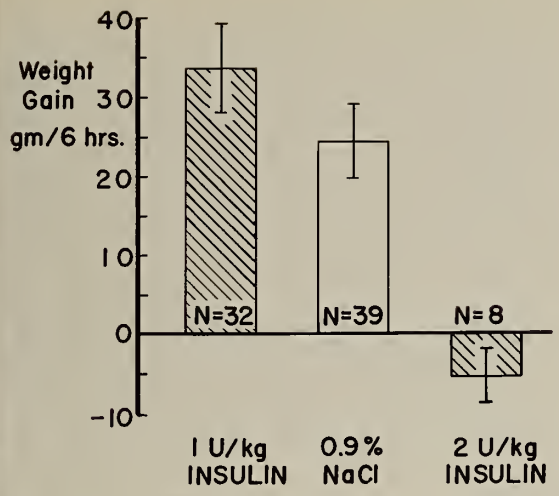


Figure 6–13. The effect of insulin on weight gain of suckling pigs. N is the number of pigs. The pigs were injected i. p. with 1.0 or 2.0 units/kg regular insular (cross hatched columns) or with an equivalent volume of 0.9% NaC1 (open column).

34±6 g/6 hr. This difference is not statistically significant, indicating that insulin does not stimulate intake in the neonatal pig although it does in the more mature pig. Two units/kg insulin caused a weight loss of 5±4 g/6 hr. Apparently 2.0 unit/kg is too high a dose for the newborn pig as it is for the weanling-age pig.

The newborn pig is not the only neonate that fails to eat in response to glucoprivation. Newborn rats also fail to increase weight gain or intake in response to insulin until they are 4 weeks of age.[16] Similarly, neonatal rats do not increase their intake in response to glucoprivation induced by the glucose analogue 2-deoxy-D-glucose.[9]

Pigs have a complicated feeding behavior pattern that may interfer with any experimental attempts to stimulate an increase in intake. The sow indicates her readiness to nurse by lying down and grunting in a slow rhythmic manner. The piglets, which are usually asleep, arouse and begin to squeal. They continue to squeal as they find their way to the sow and begin to suckle. The noises of one litter nursing arouses the sucklings in nearby pens and they too begin to suckle. This phenomenon might be considered to be social facilitation of eating. As a result, if one litter in the barn is nursing, all the other litters are also nursing. It may be difficult to stimulate intake in the suckling pig without the sow's cooperation.

In order to determine whether milk intake could be stimulated under these circumstances, we decided to use one of the simplest methods to stimulate intake: food deprivation. Half of each litter of pigs was weighed and placed in a separate pen without the sow. At the end of three hours they were reweighed to determine their weight loss during the fast. The other half of the litter remained with the sow, but they were also weighed at the beginning and end of the 3 hour period in order to determine their weight gain. At the end of the fast period all pigs were returned to the sow and allowed to suckle. At the end of a three hour refeeding period they were again weighed. Figure 6–14 shows the results. Pigs fasted for 3 hours subsequently gained 68±6 g in the 3 hour refeeding period, while their littermates who had not been fasted gained 22±6 g during the refeeding period ($t=5.35$; $p<0.001$). Apparently an increase in food intake can be stimulated by food deprivation in neonatal pigs.

In the 3 hour refeeding period the pigs which had been previously fasted gained three times as much as their nonfasted littermates. Despite their

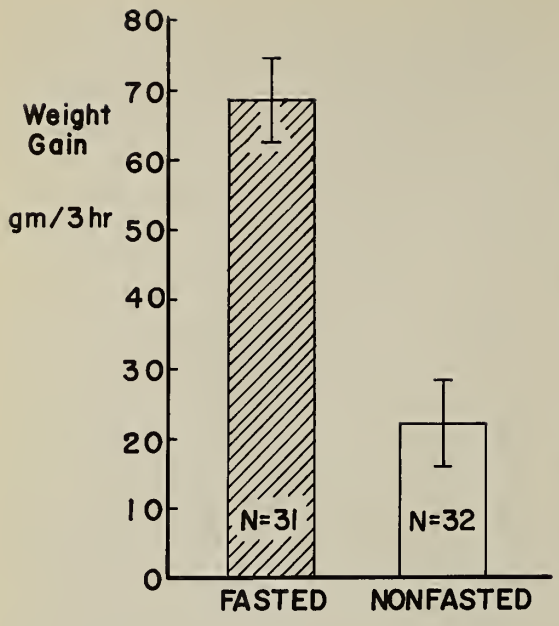


Figure 6–14. The effect of a 3 hour fast on subsequent weight gain of suckling pigs. N is the number of pigs. The fasted pigs were separated from the sow for three hours while their nonfasted littermates remained. Illustrated is the weight gain of the previously fasted piglets (cross hatched column) and in non-fasted piglets (open column) in the 3 hour refeeding period.

large gain during the 3 hour refeeding period, the fasted pigs did not completely compensate for their weight loss during the fast. As Figure 6–15 illustrates, the algebraical sum of the fasted pigs' weight loss during the 3 hour fast and their weight gain during the 3 hour refeeding period falls short of the weight gain for nonfasted groups during 6 hours of undisturbed feeding. The fasted pigs showed a new gain of only 30± 7 g/6 hr, while the nonfasted pigs showed a gain of 52± 7 g/6 hr. Failure of fasted piglets to compensate is likely due either to the sow's limited supply of milk or to the piglet's limited gastric capacity.

To determine which factor was responsible for the failure of piglets to rapidly compensate for a short period of deprivation, we repeated the experiments on bottle-fed piglets. The piglets were removed from the sow at two days of age and fed a human infant formula (Similac, Abbott). The piglets were fasted by simply removing the bottles from the rack on the cages. Following a three hour fast the bottles of milk formula were returned to the rack for a three hour refeeding period. The pigs drank 364±50 g/3 hr in this refeeding period. Intake of nonfasted piglets was 198±47 g/3 hr in the same refeeding period. These results are similar to those obtained using weight gain as a measure of intake, that is, the previously fasted pigs ate more. When net intake is calculated for the fasted and nonfasted groups during the whole 6 hour period; the results are also similar to those seen when weight gain is measured,

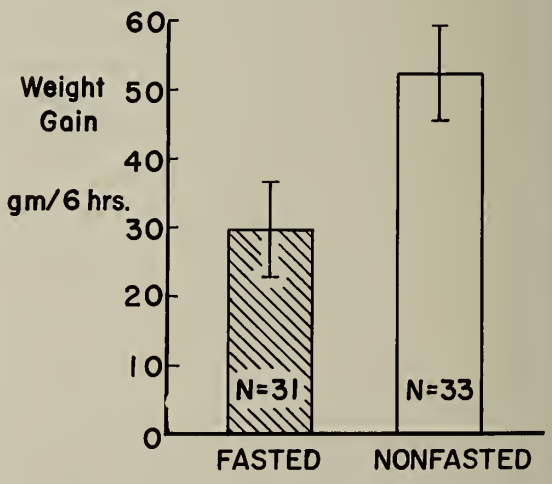


Figure 6–15. Net weight gain of suckling pigs in the combined fast and refeeding periods. N is the number of pigs. The fasted pigs (cross hatched column) lost weight during the fast period and gained weight during the refeeding period while the nonfasted pigs gained weight during both periods (open column).

that is, the net intake is smaller in the fasted piglets, they have not compensated for the fast. The net intake of the fasted piglets was 364±50 g/6 hr, while the net intake of the nonfasted piglets was 531±85 g/6 hr. Since the bottle raised piglets had access to an unlimited supply of milk, it is apparent that some factor in the piglets, perhaps gastric capacity or some other satiety signal, limits intake and prevents compensation within the 3 hour refeeding period.

The experiments using insulin to stimulate intake in neonates was also repeated in bottle-fed piglets. Following injection of 1.0 unit/kg regular insulin the pigs drank 381 g/6 hr, and following saline injection they drank 521 g/6 hr. It would appear that, although intake can be stimulated by prior deprivation in the newborn pig, it can not be stimulated by insulin-induced glucoprivation.

SUMMARY

A comparison of human and swine sweet preferences reveals marked similarities (see Table 6–I). The newborn infant [5] and the newborn pig show a marked preference for sweet fluids as do the adults of the two species. Eating in response to glucoprivation occurs in the adult pig and human.[18] Under our experimental conditions eating in response to glucoprivation does not occur in the neonatal pig. Similar studies have not been performed on human neonates, nor is it desirable to perform experiments which entail some risk on babies. The neonatal pig can serve instead as an excellant model for the development of ingestive behavior in the human infant.

TABLE 6–I

SWEET TASTE PREFERENCE AND GLUCOPRIVIC EATING IN PIG AND HUMAN

	Sweet Taste Preference	*Eating in Response to Glucoprivation*
Pig		
Neonate	present	absent
Mature	present	present
Human		
Neonate	present	unknown
Mature	present	present

REFERENCES

1. Adams, L.A.: *An Introduction to the Vertebrates.* 2nd. Edition. New York, John Wiley & Sons, 1938.
2. Book, S.A. and Bustad, L.K.: The fetal and neonatal pig in biomedical research. *J. Animal Sci.,* 38: 997–1002, 1974.
3. Bustad, L.K., McClellan, R.O., and Burns, M.P. (Eds.): *Swine in Biomedical Research.* Richland, Washington. Battelle Memorial Institute, Pacific Northwest Laboratory, 1966.
4. Chase, H.P., Marlow, R.A., Dabiere, C.S., and Welch, N.N.: Hypoglycemia and brain development. *Pediatrics,* 52: 513–520, 1973.
5. Desor, J., Maller, O., and Turner, R.: Taste in acceptance of sugars by human infants. *J. Comp. Physiol. Psychol.,* 84: 496–501, 1973.
6. Epstein, A.N. and Teitelbaum, P.: Specific loss of the hypoglycemic control of feeding in recovered lateral rats. *Am. J. Physiol.,* 213, 1159–1167, 1967.
7. Foster, R.: The reward value of saccharin solution prior to eating experience. *Psychon. Sci.,* 10: 83–84, 1968.
8. Glauser, E.M.: Advantages of piglets as experimental animals in pediatric research. *Exp. Med. Surg.,* 24: 181–190, 1966.

9. Houpt, K.A. and Epstein, A.N.: Ontogeny of controls of food intake in the rat: GI fill and glucoprivation. *Am. J. Physiol.,* 225: 58–66, 1973.
10. Houpt, T.R.: Stimulation of food intake in ruminants by 2-deoxy-D-glucose and insulin. *Am. J. Physiol.* 227: 161–167, 1974.
11. Jump, E.B., and Weaver, M.E.: The miniature pig in dental research. In Bustad, L.K., McClellan, R.O., and Burns, M.P. (Eds.) *Swine in Biomedical Research.* Richland, Washington, Battelle Memorial Institute, Pacific Northwest Laboratory, 1966, pp. 543–556.
12. Kare, M.R.: Comparative aspects of the sense of taste. In Kare, M.R. and Halpern, B.P. (Eds.) *Physiological and Behavioral Aspects of Taste,* Chicago, University of Chicago Press, 1961, pp. 6–15.
13. Kare, M.R., Pond, W.G., and Campbell, J.: Observations on the taste reactions in pigs. *Animal Behav.,* 13: 265–269, 1965.
14. Kelly, M., Butler, D.G., and Hamilton, J.R.: Transmissible gastroenteritis in piglets: A model of infantile viral diarrhea. *J. Pediatrics,* 80: 925–931, 1972.
15. Kennedy, J.M. and Baldwin, B.A.: Taste preferences in pigs for nutritive and nonnutritive sweet solutions. *Animal Behav.,* 20: 706–718, 1972.
16. Lytle, L.D., Moorcroft, W.H., and Campbell, B.A: Ontogeny of amphetamine anorexia and insulin hyperphagia in the rat. *J. Comp. Physiol. Psych.,* 77: 388–393, 1971.
17. Mackay, E.M., Calloway, J.W., and Barnes, R.H.: Hyperalimentation in normal animals produced by protamine insulin. *J. Nutr.,* 20: 59–66, 1940.
18. Mayer, J.: Regulation of energy intake and the body weight: The glucostatic theory and the lipostatic hypothesis. *Ann. N.Y. Acad. Sci.,* 63: 15–43, 1955.
19. Mersman, H.J.: Metabolic patterns in the neonatal swine. *J. Animal Sci.,* 38: 1002–1030, 1974.
20. Mount, L.E.: The thermal insulation of the newborn pig. *J. Physiol.,* 168: 698–705, 1963.
21. Pekas, J.C., Combs, G.E., Vanderpopuliere, J.M., and Wallance, H.D.: Hypoglycemic compounds as appetite stimulants for baby pigs. *J. Animal Sci.* 18: 1282–1291, 1959.
22. Sampson, J., Hester, H.R., and Graham, R.: Studies on baby pig mortality. II. Further observations on acute hypoglycemia in newly born pigs (so-called baby-pig disease). *J. Am. Vet. Med. Assoc.,* 100: 33–37, 1942.
23. Shelley, H.J. and Neligan, G.A.: Neonatal hypoglycemia. *Brit. Med. Bull.,* 22: 34–39, 1966.
24. Sisson, S. and Grossman, J.D.: *The Anatomy of the Domestic Animals.* 4th. Edition. Philadelphia, W. B. Saunders Co. 1953.
25. Wilson, M.R.: Immunologic development of the neonatal pig. *J. Animal Sci.,* 38: 1018–1021, 1974.

Chapter 7

COMPARATIVE OBSERVATIONS ON THE ONTOGENY OF TASTE PREFERENCE

Harry L. Jacobs, Edwin R. Smutz and Cynthia N. DuBose

Our interest in the ontogeny of taste preference started from an attempt to evaluate the oft-claimed generality of the "sweet-tooth" across species (see Pfaffmann [17] in this volume). In order to minimize post ingestion effects and to isolate sensory factors in preference, we chose to use a brief exposure technique, applying drops of test solution to the tongue and observing oral and facial responses normally associated with consummatory behavior.

ONTOGENY OF TASTE PREFERENCE IN A HUMAN NEONATE

The initial observations were carried out in 1960 on a human neonate, in a hospital nursery from birth to six days of age, and at home at ten and thirty days of age.* Table 7–I shows the responses associated with acceptance and rejection in this study. On most trials more than one specific behavior could be observed. Quite often, the initial behavior was a continuation of the response pattern to previous solutions. Thus, it was necessary to observe the subject for up to 30 seconds while repeatedly applying drops of solution to the tongue, in order to define a consistent response pattern in a single trial.†

Some examples from the original data sheets will illustrate this point. (A) Quinine (first trial of test, 45 minutes post-prandial)—"almost asleep, frown, grimace, purse, turns head away." (B) Quinine (after sucrose)—"starts to accept, grimace, purse, close mouth, spit out." (C) Water (after sucrose)—"suck, holds still, open mouth, lets leak out." (D) Saccharin (after water)—"lick, suck, moves mouth to follow catheter as it is withdrawn."

Table 7–II presents the relative

*This study was carried out at Strong Memorial Hospital, Rochester, N.Y., where the subject, Emily Jacobs, was born on February 11, 1960. Birth was full term, with routine delivery. Weight was 3000 g., length was 49 cm. Hospital formula, 5% sucrose solution containing 1/3 evaporated milk and 2/3 water, was administered at 4 hour intervals. At home, she was bottle-fed on a self-demand schedule over the month that she was observed. Number of feedings varied from 4–7 per day. Single daily feedings of cereal in addition to formula began at three weeks of age. Seventeen test sessions were run between 0 and 6 days of age in the hospital nursery. Two additional sessions were run, on days 10 and 30 at home. Ten of the seventeen test sessions were run blind with coded solutions available to the experimenter. These included tests 6–15, starting at two days of age in the nursery.

†Each test consisted of 10–30 tongue applications of from 4–8 stimulus solutions administered with a PE–90 catheter mounted on a 5 cc syringe. Solutions were administered in a predetermined random order. The catheter was laid on the tongue and single drops of solution were applied until a consistent response was observed. Each trial lasted from 10–30 seconds, during which 1–1.5 cc were administered. At least 30 seconds elapsed between applications.

TABLE 7–I

BEHAVIOR CATEGORIES DEVELOPED IN A STUDY OF TASTE PREFERENCES IN A HUMAN NEONATE [1]

General Response	*Specific Behavior*	*Preference*
Active	Turn to Stimulus Swallow Suck Lick Pucker	Acceptance
Passive	Open Mouth Leak Out Drip	Neutral
Active	Whimper Frown Grimace Purse Spit Cry Retrusion Turn from Stimulus	Rejection

Note: Subject, female neonate; 17 test sessions 0–6 days of age, one test session each at 10 and 30 days of age.

TABLE 7–II

RELATIVE ACCEPTABILITY OF TEST SOLUTIONS IN A STUDY OF TASTE PREFERENCES IN A HUMAN NEONATE

Rankings of Test Solutions		
0–6 Days Old (Hospital)	*10 Days Old (Home)*	*30 Days Old (Home)*
.1% Saccharin/10% Sucrose	.1% Saccharin	.1% Saccharin
20% Lactose		
Cow's Milk/13.5% Glucose	Cow's Milk	Cow's Milk
Water/7% Lactose	Water	Water
.85% NaCl		
Human Breast Milk		
.0004M QSO_4	.0004M QSO_4	.0004M QSO_4

Note: Subject, female neonate; 17 test sessions 0–6 days of age, one test session each at 10 and 30 days of age.

Rankings are based upon the frequency of occurrence of specific behaviors that fitted preference categories as shown in Table 7–I. The paired items could not be differentiated by this technique.

Saccharin, sucrose, water, and quinine sulfate, >50 applications each; lactose (20% and 7%), cow's milk, and sodium chloride, >20 applications each. Glucose and human breast milk, 4 applications on last day in hospital. Human breast milk obtained from voluntary donor 5 hours before testing; heat sterilized in boiling water.

rankings of the test solutions used in this study. The specific responses were recorded and later converted into five qualitative categories, using plus (+) and minus (−) notations. The categories were active acceptance (++), passive acceptance (+), neutral (+−), passive rejection (−), and active rejection (−−). These were later arranged in a Trial X Solution matrix. The solutions were then ranked by tabulating the frequency of occurrence of the above categories across trials and test sessions.

The results were quite clear in showing neonatal sweet acceptance (.1% [.00546 M] saccharin, 10% [.292 M] sucrose) and bitter rejection (.0004 M QSO_4), with water consistently neutral. These responses appeared during

the first day of life. Over the first six test sessions at 1, 10, 12, 21, 32, and 37 hours after birth, the rankings were consistent, with saccharin (15 presentations) = sucrose (12 presentations) > water (12 presentations) > quinine (13 presentations).

Comparison with adult human taste data from the work of Cameron [1] and Moskowitz [14, 15] shows that the results from our tests using sweet solutions are consistent with the hypothesis of a monotonic relationship between sweetness intensity and acceptability within the concentration range we tested. An exception is 20% (.584 M) lactose, which is about equal in sweetness to 10% sucrose, yet less acceptable in our tests.

It is interesting to note that, although cow's milk showed mild acceptance distinguishable from water, the four samples of human breast milk showed consistent rejection. The preference for cow's milk is consistent with its high acceptability in young adults, as shown by Meiselman, et. al. in 1974.[11] The rejection of human breast milk was not anticipated.* However, our method of tongue application limited sensory input to simple taste stimuli, while normal breast feeding combines flavor with warmth, movement, and tactual stimulation during nurturance.

At this point, we modified our measurement techniques to make them applicable to the rat † and began a series of observations on rat neonates.

ONTOGENY OF TASTE PREFERENCES IN NEONATE RATS

Figure 7–1 summarizes the results of our initial studies on neonate rats.[5,6,7] These studies traced the development of taste preferences from birth through weaning. As in the human neonate, sweet substances were highly accepted, water was neutral and not distinguishable from mildly sweet sugars (2.8% [.082 M] lactose, 4.0% [.117 M] lactose), and quinine was strongly rejected. Since 2.8% lactose, equal in concentration to that found in rat's milk, was not distinguishable from water, neonatal saccharin preference could not be explained by a simple conditioning paradigm, in which the sweet taste of rat's milk is associated with hunger reduction each time the neonate is fed. We concluded that the neonate demonstrates a very strong innate preference, based upon taste alone.[7]

The rest of this paper will address further methodological and substantive issues arising from consideration of the data presented in Figure 7–1.

*Since our data on sugar solutions indicate that a sweeter solution is preferred to a less sweet solution, on the basis of sweetness alone, we would have expected that human breast milk (7.0% lactose) would be more acceptable than cow's milk (4.8% lactose). However, it should be pointed out that milk is a complex mixture composed of many components, including sugar, which produce its flavor.

†The techniques used in rats have been described elsewhere.[5,6,7] The major differences between working with the rat and the human neonate were: (A) We held the rat in one hand within six inches of a 60 W bulb for warmth during testing, while the human neonate was allowed to lie on her back in her nursery crib, her body under the crib blanket and sheet, and her head free. (B) We scaled down the solution application technique to the rat neonate by using one inch of PE–10 tubing mounted on a 2 cc syringe. (C) We developed a 4-point scale ranging from maximum negative response (1.0) to maximum positive response (4.0) which was quite similar to the human behavior categories shown in Table 7–I without reference to facial expressions or emotional status; that is, "whimper," "frown," "grimace," "spit," and "cry" were omitted.

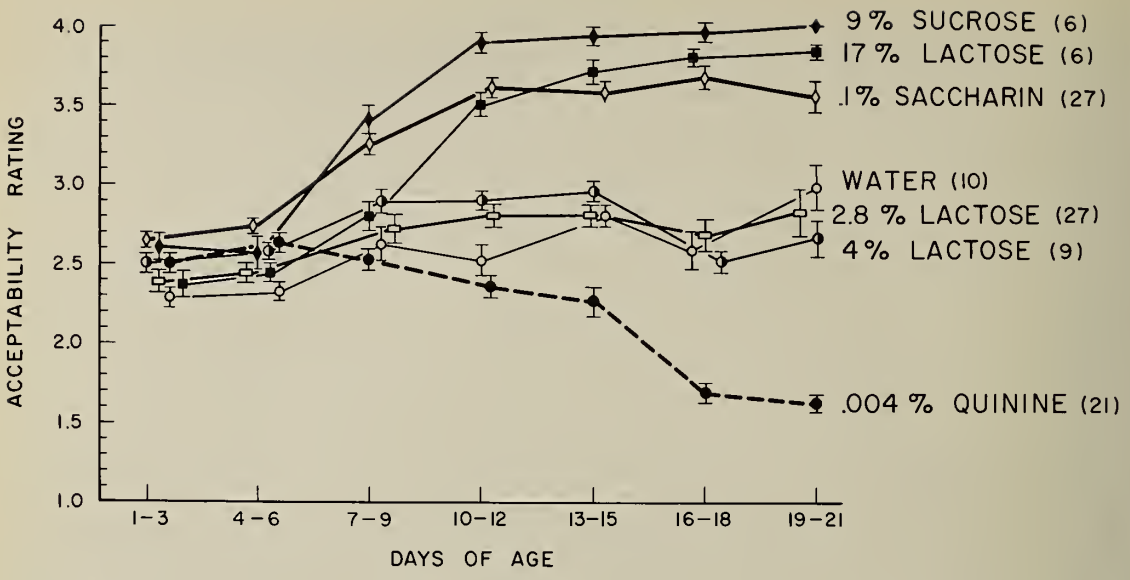


Figure 7–1. Ontogeny of sweet preference and bitter avoidance in preweanling rats. Parentheses enclose number of subjects tested. Four percent lactose condition added to data originally presented by Jacobs and Sharma in 1969.[7]

Neonatal test experience and acceptability

What of the gradual development of sweet preference and quinine rejection over the 21 days of testing shown in Figure 7–1? To what extent are the shapes of the curves due to repeated experience with the testing procedure? This was evaluated by presenting quinine to naive rats at 15 days of age, and water and saccharin at 19 days of age. Maximum saccharin preference and quinine rejection were not dependent upon practice in taste tests during the first days of life (see Figure 7–2). Thus, the curves in Figures 7–1 and 2 are "maturation" curves. What is it that is maturing? We have suggested that the delays in showing maximum responses are due to the need for maturation of the motor skills required to show acceptance or rejection in our acceptability tests.[7] Mistretta,[13] in this volume and elsewhere,[12] suggests a more interesting possibility, that these delays are correlated with differential maturation of the taste buds necessary to discriminate these stimuli. Thus, the relatively early occurrence of sweet acceptance may be correlated with the early development of taste buds on the anterior surface of the tongue, and the relatively late occurrence of bitter rejection may be correlated with the late development of taste buds on the posterior tongue surface.

Extent of individual and temporal differences

The rather smooth ontogeny curves shown in Figure 7–1 are based upon data collected 2–4 times daily on groups of rats and are plotted on the basis of three-day averages. To what extent is the course of development misrepresented by averaging data across days and subjects? It can be

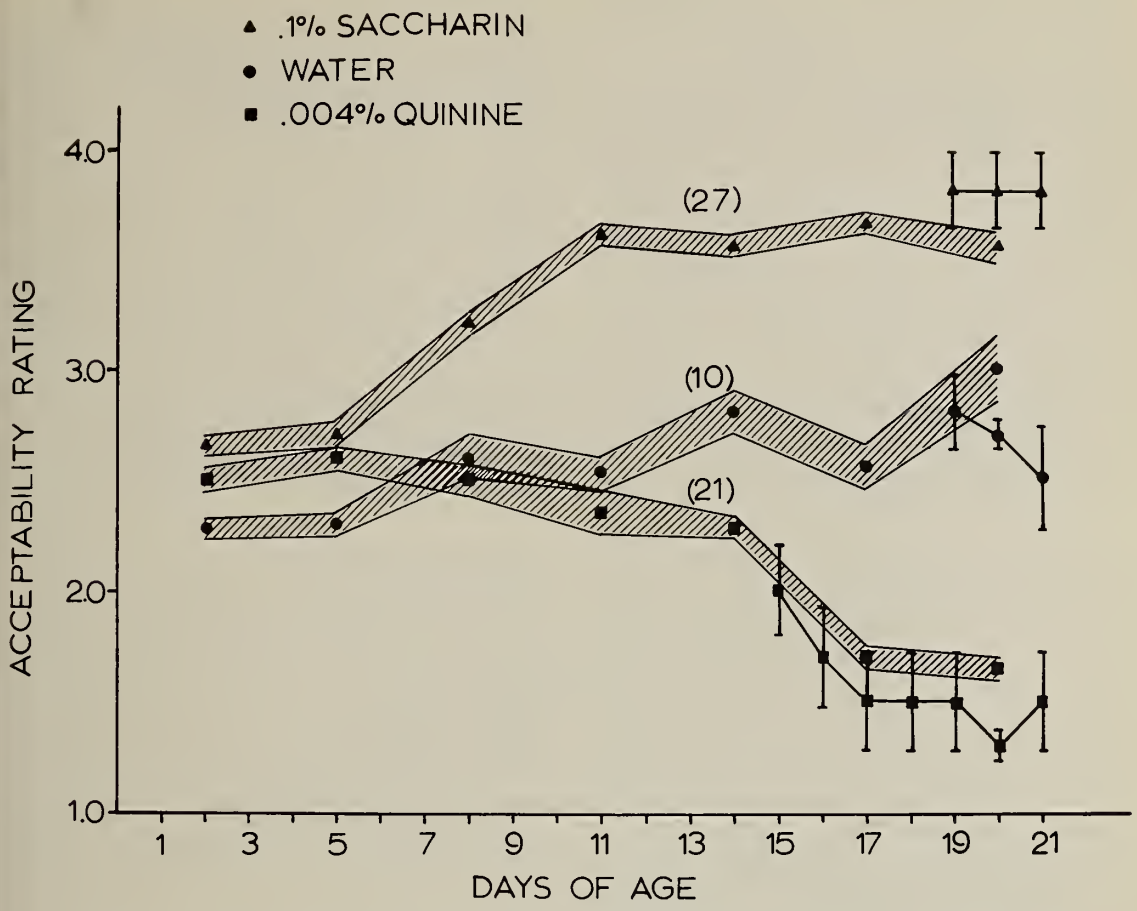


Figure 7–2. Preweanling acceptability values with and without neonatal test experience. The shaded curves show the gradual development of saccharin preference and quinine avoidance with daily tests from birth to weaning at 21 days of age. The shaded areas span the mean ±SE and are plotted by three day average scores. Parentheses enclose the number of subjects tested. The singleline curves plotted daily show mean ±SE from a litter of six rats first tested with quinine sulfate at day 15, and with water and saccharin at day 19. This group shows equal acceptability levels *without* the opportunity to repeatedly taste these stimuli from birth.

seen that the variability around the daily means in Figure 7–2 is greater than that around the three-day means in that Figure. A similar difference is also seen in Figure 7–3A where three-day averaging of one litter's data produces a smoother less variable ontogeny curve than daily averaging. Figure 7–3B illustrates the individual variability seen among littermates when their data are plotted in three-day sets. These differences appear even greater in daily plots.

In all our studies, the extent of variability is the same order of magnitude as in the cases shown in Figure 7–3. Thus, our conclusions are not biased by the averaging methods chosen for graphical and statistical presentation. Were we interested in

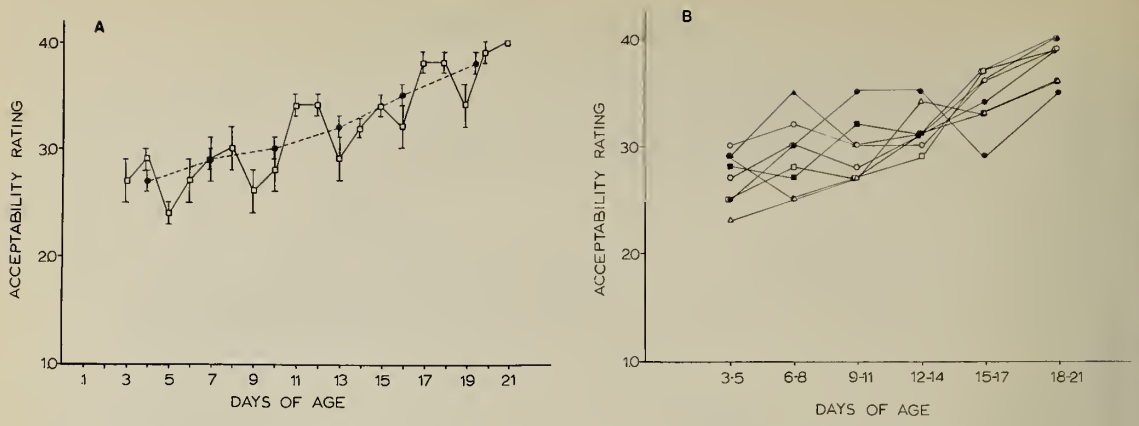


Figure 7–3. Sources of variability in plots of acceptability scores for one eight pup litter of rats tested 2–3 times daily with .1% saccharin. The solid line curve in plot A connects the litter's daily means. The dashed line curve connects means for blocks of 3 days. Brackets show ±SE. Curves on plot B connect three day means for individual pups.

empirical or rational equations to describe the data, these considerations would have become much more important. Over-generalizing from the specific shape of growth functions based upon averaged data is a classic problem applicable to all developmental studies, of course, and is more easily recognized than resolved. In these studies of neonate rats there is one source of individual variability that could be reduced, at least in principle. The variability in individual maturation rates of the responses we chose to measure could be better controlled by using body weight and growth rate, instead of age in days, to match subjects.

Effect of scale on acceptability measures

The data in Figures 7–1 through 3 were collected using a four-category scale. Was the shape of the developmental function an artifact of the number of scale categories? Figure 7–4 compares the four-category scale with one allowing the use of three additional categories, 1.5, 2.5, and 3.5, in determining each acceptability rating. The seven-category scale gave consistently lower ratings throughout. However, as in the case of the individual differences discussed above, the acceptance trends using the seven-category scale were the same as those using the four-category scale,

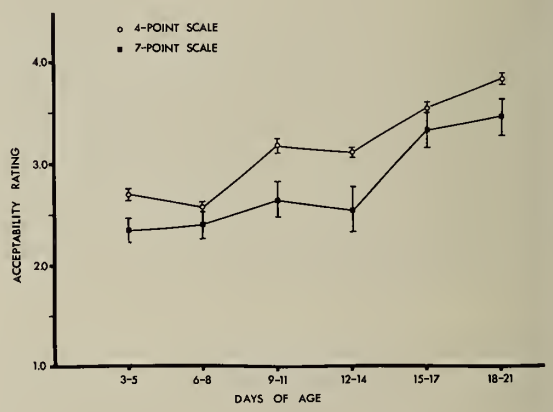


Figure 7–4. Effecf of number of scale categories on measurement of .1% sodium saccharin acceptability in rats tested 2–3 times daily. Three litters (N=24) given 4-point scale started on day three, the single litter (N=6) given 7-point scale on day two. Data from day two are omitted. Means ±SE are shown.

and our conclusions about the solutions tested in Figures 7–1 though 3 would not have been changed significantly had the seven-category scale been used.

Perhaps a more extensive scale comparison using a within-subjects design would provide a more definitive evaluation of the potential usefulness of the scale with the larger number of categories. However, in the absence of obvious advantages for either scale, the choice can be made on the basis of the experimenter's preference.

Discrimination within taste qualities

How specific is acceptance or rejection within taste qualities in neonates? Figure 7–5 directly compares two sweet (pure saccharin and sodium saccharin) and three bitter (quinine hydrochloride, quinine sulfate, and sucrose octa-acetate) solutions. The animals failed to discriminate among solutions of the same taste quality, consistently showing high acceptance of all sweet solutions and high rejection of all bitter solutions throughout the testing period.

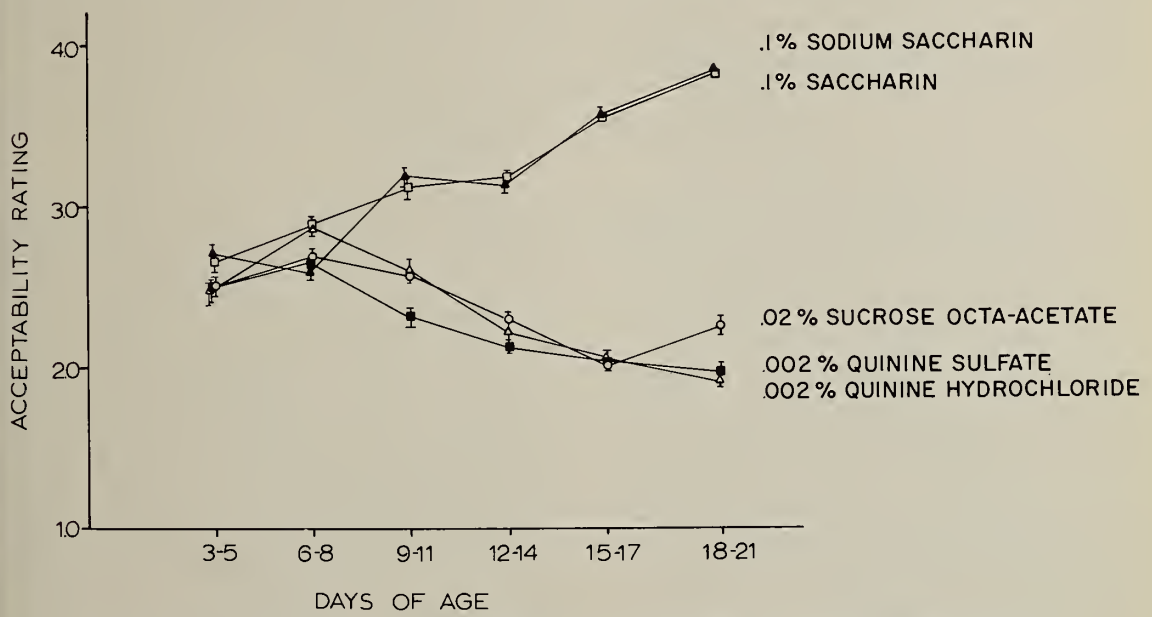


Figure 7–5. Ontogeny of sweet preference and bitter avoidance across specific stimulus solutions. Three litters (24 subjects) run on two sweet and three bitter stimuli from days 3–21. Means ±SE are shown.

COMPARATIVE ACCEPTABILITY—NEONATE HAMSTERS

Detailed observations of one human neonate and a series of studies on neonate rats showed similar responses to sweet and bitter solutions (Table 7–II, Figure 7–1). To expand our explorations to other species, we applied the technique developed for rats to three litters of Golden Hamsters. Figure 7–6 compares the responses of hamsters to bitter, neutral, and sweet stimuli to those demonstrated by rats of identical chronological age. The results are presented for days 19—21 after birth. Both groups were presented quinine sulfate, quinine hydrochloride, water,

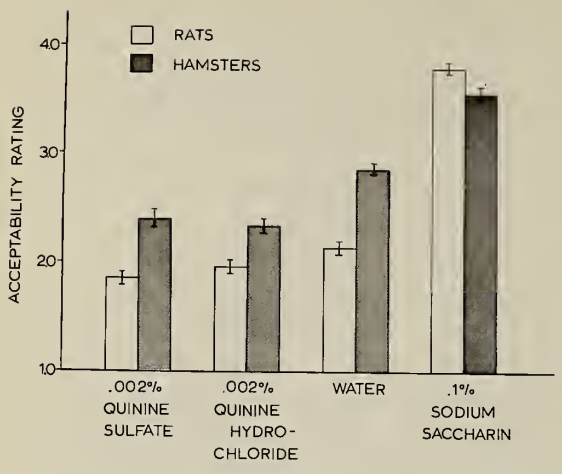


Figure 7–6. Comparisons of sacharin preference and quinine avoidance in rats and hamsters. Rats (N=24) tested 2–3 times per day; hamsters (N=18) tested once per day. Data shown are for last three-day block in animals tested during first 21 days of life. Three-day mean ±SE of days 19–21 for both groups.

and saccharin. Although quinine and water were significantly more acceptable to the hamsters than to the rats and saccharin was less acceptable, the relative preferences were the same in that saccharin was more acceptable than water, which was more acceptable than quinine.

SUMMARY AND COMMENT

A category scale of taste preference was developed during a series of observations on a human neonate. The scale was based upon the oral behavior of the subject toward solutions applied to the tongue in a brief exposure test. This technique was then modified and applied to rats in studies of the ontogeny of acceptance and rejection, gathering descriptive data and asking methodological questions. Finally, it was used on hamsters for comparative purposes.

The results on the human neonate, rats, and hamsters were similar, showing sweet acceptance and bitter rejection, saccharin > water > quinine. Responses to milk sugar (lactose) in the human neonate and rats suggested an innate positive hedonic response to sweetness rather than learned preference.[7] With this technique, the human neonate showed mild acceptance of cow's milk and mild rejection of human breast milk.

Methodological studies in rats showed (1) repeated experience with test solutions was unnecessary for the demonstration of a differential response, (2) individual differences should caution investigators against interpreting the relatively smooth, averaged ontogeny functions as more than a general trend useful for comparing the preferences for different taste stimuli, (3) a four-point acceptability scale was adequate to discriminate among taste qualities, (4) rats responded to sweet or bitter stimuli as a class, failing to discriminate among various sweet solutions or among different bitter solutions.

The brief exposure technique developed in these experiments and the solutions chosen for test were based upon the published literature on taste preferences in adult rats; therefore, our results can be related to electrophysiological and behavioral investigations of the chemical senses

and taste preferences in animals (see Pfaffmann,[17] Richter,[18] Young,[21] Le Magnen [9] in this volume). Applying closely analogous techniques in animal and human taste studies, we have obtained strikingly similar results that should encourage others toward cross-species comparison. Furthermore, we would anticipate that contributions to the understanding of the ontogeny of human taste preferences would come from comparison of the measures used in our acceptability scale and the oral reflex and ingestion techniques that have been used on human neonates by others (see Crook,[2] Desor, et. al.[3] Engen,[4] Johnson and Salisbury,[8] Lipsitt,[10] Nowlis,[16] Steiner,[19] and Weiffenbach [20] in this volume).

REFERENCES

1. Cameron, A.T.: The taste sense and the relative sweetness of sugar and other sweet substances. *Scientific Report Series—Sugar Research Foundation,* No. 9, 1–72, 1947.
2. Crook, C.: Taste and the temporal organization of neonatal sucking. This volume.
3. Desor, J., Maller, O., and Greene, L.: Preference for sweet in humans: infants, children and adults. This volume.
4. Engen, T.: The discrimination of dextrose and water by newborn infants. This volume.
5. Jacobs, H.L.: Observations on the ontogeny of saccharin preference in the neonate rat. *Psychonomic Science,* 1:105–106, 1964.
6. Jacobs, H.L.: Taste and the role of experience in the regulation of food intake. In Kare, M., and Maller, O. (Eds.), *Chemical Senses and Nutrition.* Baltimore, Johns Hopkins Press, 1966, pp. 187–200.
7. Jacobs, H.L. and Sharma, K.N.: Taste vs. calories: Sensory and metabolic signals in the control of food intake. In Morgane, J.P., (Ed.), *Neural Regulation of Food and Water Intake. Annals N.Y. Acad. Sci.,* 157: 1084–1125, 1969.
8. Johnson, P. and Salisbury, D.M.: Preliminary studies on feeding and breathing in the newborn. This volume.
9. LeMagnen, J.: Sweet preference and the sensory control of caloric intake. This volume.
10. Lipsitt, L.: Taste in human neonates: its effects on sucking and heart rate. This volume.
11. Meiselman, H.L., Waterman, D., and Symington, L.E.: Armed Forces Food Preferences. United States Army Natick Development Center Technical Report 75–63–FSL, December 1974.
12. Mistretta, C.: Topographical and histological study of the developing rat tongue, palate and taste buds. In Bosma, J.F., (Ed), *Third Symposium on Oral Sensation and Perception.* Springfield, Thomas, 1972, pp. 163–187.
13. Mistretta, C.M., and Bradley, R.M.: Taste *in utero:* theoretical considerations. This volume.
14. Moskowitz, H.R.: Sweetness and intensity of artificial sweeteners. *Psychonomic Science,* 8:40–42, 1970.
15. Moskowitz, H.R.: The sweetness and pleasantness of sugars. *American Journal of Psychology,* 84:387–405, 1971.
16. Nowlis, G.: From reflex to representation: taste-elicited tongue movements in the human newborn. This volume.
17. Pfaffmann, C.: Biological and behavioral substrates of the sweet tooth. This volume.
18. Richter, C.P.: Six common sugars as tools for the study of appetite for sugar. This volume.
19. Steiner, J.: Facial expressions of the neonate infant indicating the hedonics of food related chemical stimuli. This volume.
20. Weiffenbach, J.M.: Sensory mechanisms of the newborn's tongue. This volume.
21. Young, P.T.: Role of hedonic processes in the development of sweet taste preferences. This volume.

PART III
SWEET SENSITIVITY AND PREFERENCE IN HUMAN NEWBORNS

Chapter 8

PRELIMINARY STUDIES ON FEEDING AND BREATHING IN THE NEWBORN

P. Johnson and D.M. Salisbury

The attempt to relate the nature of a newborn's feed to its effect on patterns of sucking and breathing originated from our studies of the onset and control of effective breathing at birth. The upper airway, in particular the laryngeal inlet, is not only the aditus to the lower airway but also to the gut. The converse consideration was emphasized by Bosma.[3]

For the last two years, we have been examining the physiological function of the airway defense mechanisms in animals. Proceeding in a perhaps unusual way, we introduced fluids retrograde from the trachea to the pharynx. The animal investigations, carried out chiefly in the lamb, but also with newborn monkey, calf and foal and with mature sheep and monkeys are briefly described, since they led to the human studies.

ANIMAL STUDIES

Fetuses

Five years ago, Dawes and his associates in the Nuffield Institute made observations on chronically monitored healthy fetal lambs showing that breathing activity was a normal activity of fetal life.[5] Figure 8–1 reproduces a 2 hour long record from a healthy lamb fetus 7 days after surgery. Electrocortical activity is measured from bilateral stainless steel screws on the dura, tracheal pressure by catheter to a pressure transducer and tracheal flow by an electromagnetic flowmeter inserted in the trachea. An important feature of this record is the occurrence of periodic rapid irregular breathing activity at the time of low voltage cortical activity, alternating with periods of apnea and high voltage cortical activity. This finding of periodic breathing activity in the healthy fetus has changed our view of postnatal breathing.[1] Another feature of fetal breathing activity is particularly germane to the comments that Dr. Mistretta made yesterday [11] and should be emphasized. Most of the stimuli that both stimulate and depress respiration after birth, are similarly potent stimuli during fetal life. Their potency for the fetus is, however, limited almost exclusively to the periods of rapid low voltage electrocortical activity.[2] During periods of high voltage electrocortical activity, rapid breathing activity is seldom observed and the fetuses' respiration is relatively unresponsive to stimulation.

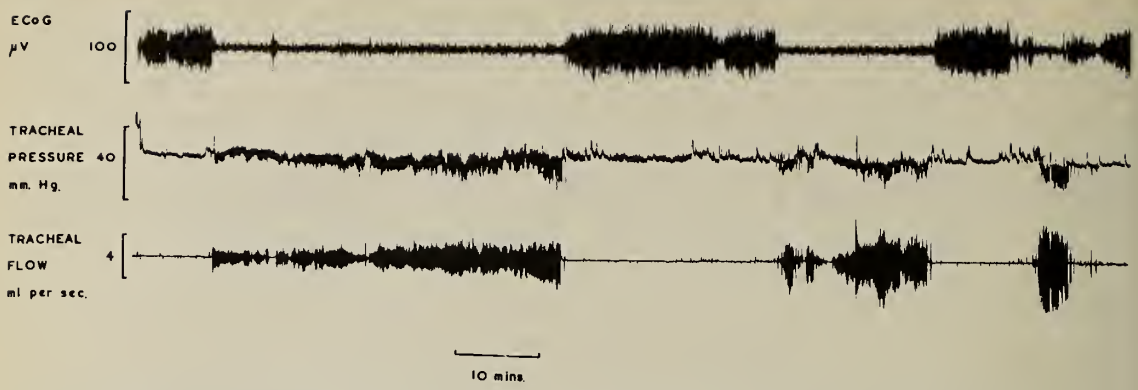


Figure 8–1. Electrocorticogram (ECoG), tracheal pressure and tracheal flow in a chronically tracheotomized lamb fetus. See text for details of preparation and comment.

Similar factors may influence sucking and swallowing.

Newborns

In our experiments, we observed that different fluids in the upper airway elicited reproducibly different rates of swallowing. Since the newborn lambs can often be studied in the unanaesthetised state observations on the lamb can play an important role in relating experimental observations to normal function.

Method. The experimental preparation which formed the basis of the neonatal observations in animals can be seen in Figure 8–2. The trachea is simply divided permitting the animal to breathe through the caudal end, and selected fluids could be introduced into the upper airway.

Ninety three lambs ranging from birth to 3 months and of varying breeds have been used in these studies. Usually the older lambs were under light general anaesthesia (sodium pentabarbitone) with local anaesthetic (1% Xylocaine) infiltrated into the areas requiring surgery. Electrocortical activity was recorded from biparietal steel screws fixed to the skull and insulated from it by dental acrylic. The screw tips rested on the dura. Breathing was recorded either quantitatively by a Fleish pneumotachograph when a tracheotomy was performed, or qualitatively by tidal CO_2 or impedance pneumography (chest electrodes) when the airway was intact. In some animals a continuously recording, rapid response catheter tip oxygen electrode was used, particularly in an effort to distinguish primary from secondary reflex respiratory events. Swallowing was recorded by a cannulated electromagnetic flow meter inserted into the upper end of the oesophagus, by a tensionmeter attached to the thyroid cartilage or a fluid filled pharyngoesophageal catheter. More recently recordings from appropriate chronically implanted EMG electrodes have been employed to index swallowing. Blood pressure and heart rate were measured from arterial catheters connected to a Bell and Howell pressure transducer. Single unit analysis of the superior laryngeal nerve (SLN) was carried out by dividing portions of

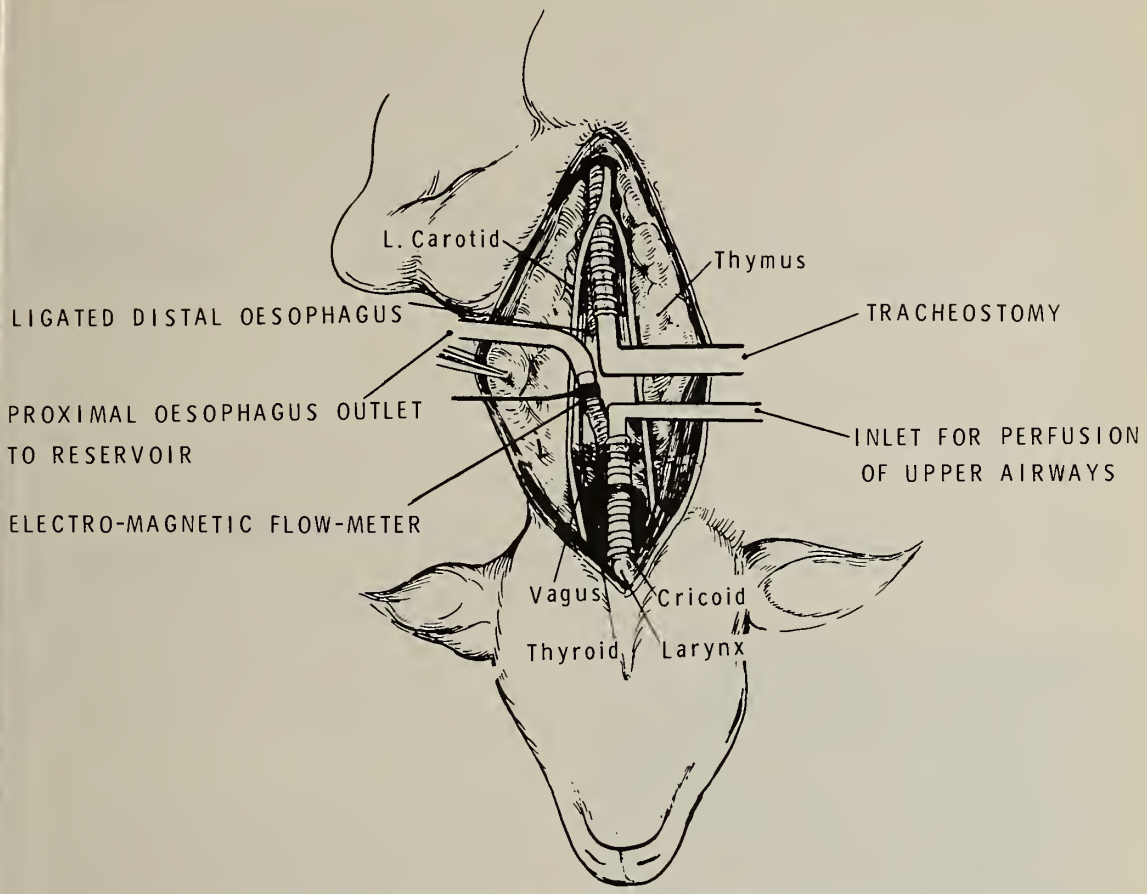


Figure 8–2. Preparation employed in study of upper respiratory influences upon fetal and neonatal respiration. See text for further details.

one or both nerves and placing fine strands on an electrode under paraffin oil. Often the contralateral pathway can be maintained intact and receptor/fibre characteristics related to simultaneously observed swallowing and cardiorespiratory responses. Clearly, not all procedures were applied to each animal. Similar methods have been employed in smaller numbers of calves, dog pups, kittens and monkeys.

Results. The record reproduced in Figure 8–3 shows the characteristic response of our preparations to saline and water that has been described before.[7] The introduction of isotonic saline into the upper airway elicits swallowing and very little disturbance of breathing, heart rate or blood pressure. In contrast water, similarly introduced, causes a vigorous burst of swallowing and instant apnoea. The response is especially noticeable if the animal is awake and alert. Of considerable importance is an initial bradycardia and hypertension not simply related to the ensuing asphyxia. We have discussed the significance of this response elsewhere.[6]

For our present purposes, the important point, is the persistence of the young animal's apnoea. It continues until the stimulus is removed. Only

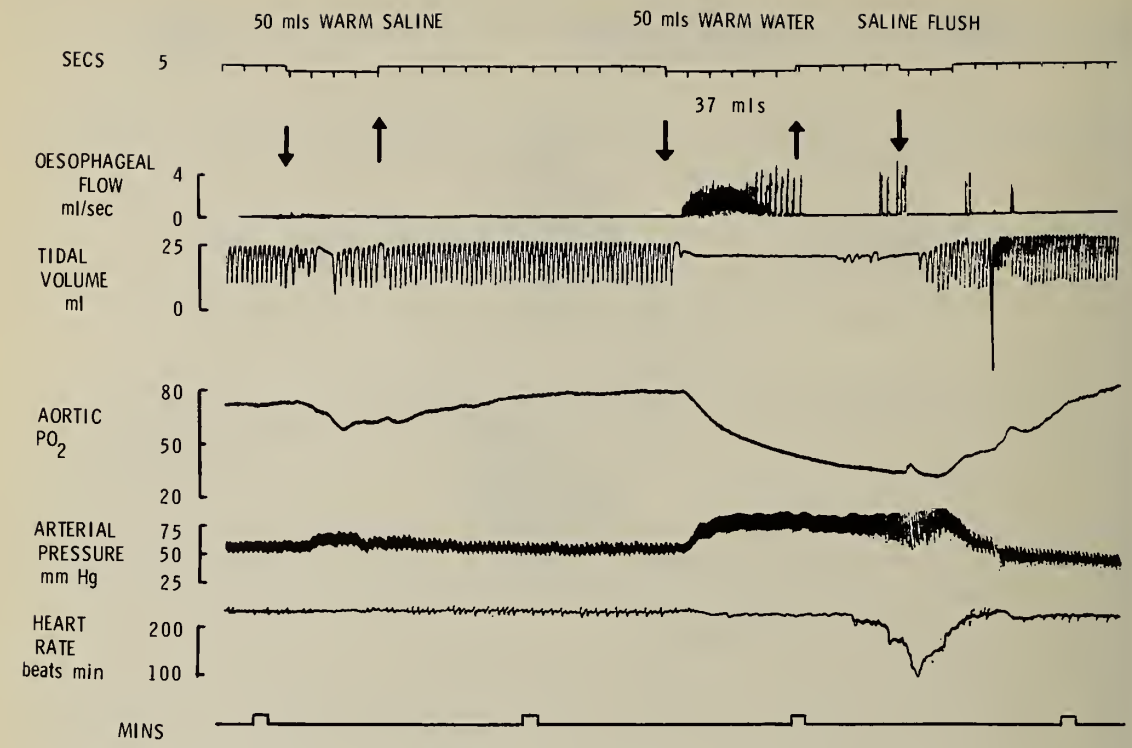


Figure 8–3. Effect upon respiration of introducing saline and water into the upper airway.

when the water stimulus is replaced by normal saline does the animal re-establish breathing.

Table 8–I summarizes the effects of perfusion of the upper airway with other fluids on breathing and swallowing.

Discussion. To what purpose does the animal distinguish between water and saline in this region of the airway? Tracheal fluid and amniotic fluid are the liquids most likely to be present in this region in fetal life, and as can be seen in Table 8–I they have no effect on breathing, and do not elicit swallowing. The next fluids, after air, that the lamb is likely to meet in postnatal life is sheep's milk, and it has little or no effect on breathing or swallowing. Cow's milk, however, caused apnoea and swallowing although the response is not as marked as that caused by water. The finding that normal saline had no effect, whereas, water caused apnoea and swallowing is the converse of results that were presented elsewhere in this volume.[11] Nonetheless they suggest a relation to taste as observed in the oral region. Certainly the presences of C. Mistretta and R. Bradley in the Institute at the time hastened this consideration.

It is not unreasonable to suggest that our results reflect a locally distinctive form of taste or chemosensitive mechanism. The distribution of extra-lingual taste buds in the human infant [9] provides a suggestive homology. Figure 8–4 is a parasagittal section through the entrance to the larynx of a 134 day (near-term) fetal lamb under low power. The laryngeal

TABLE 8–I

RESPONSE OF NEWBORN LAMBS TO PERFUSION OF UPPER AIRWAY

Perfusing Liquid	*Apnoea*	*Swallowing*
Water	+	+
Saline N	–	–
HCL 0.01 N	+	+
Quinine 0.02 M	+	+
Sucrose 1 M	+	+
Cow's milk	+	+
Sheep's milk	–	–
Amniotic fluid	–	–
Tracheal fluid	–	–

inlet and the epiglottis can be identified. The chemosensitive area is quite precisely the laryngeal surface of the epiglottis, and the arytenoid processes. Section of the superior laryngeal nerves abolishes this reflex response. Taste-bud like structures are found in the mucosa of the sensitive area just described (Fig. 8–5). There were some 1,000 taste buds in this area of an example lamb. However, there are also numerous fine free-nerve endings well seen on silver staining of similar sections and there may be other specialized nerve endings.

Regardless of the structure of the chemoreceptor, it was important to decide in studying the end organ here, whether there was a receptor in the laryngeal area that could be responsible for this prolonged apnoea in the newborn. In collaboration with A. Storey,[13] activity was recorded from single units of the uncut superior laryngeal nerve (SLN).

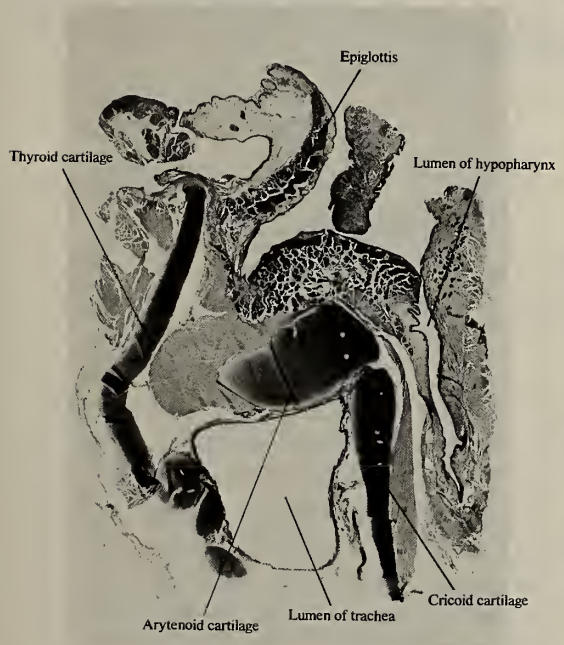


Figure 8–4. Sagittal section through the pharynx and larynx 0.5 cm lateral to the mid-line.

Figure 8–5. Histological section of arytenoid epithelium of the lamb showing a typical "taste bud" (×610).

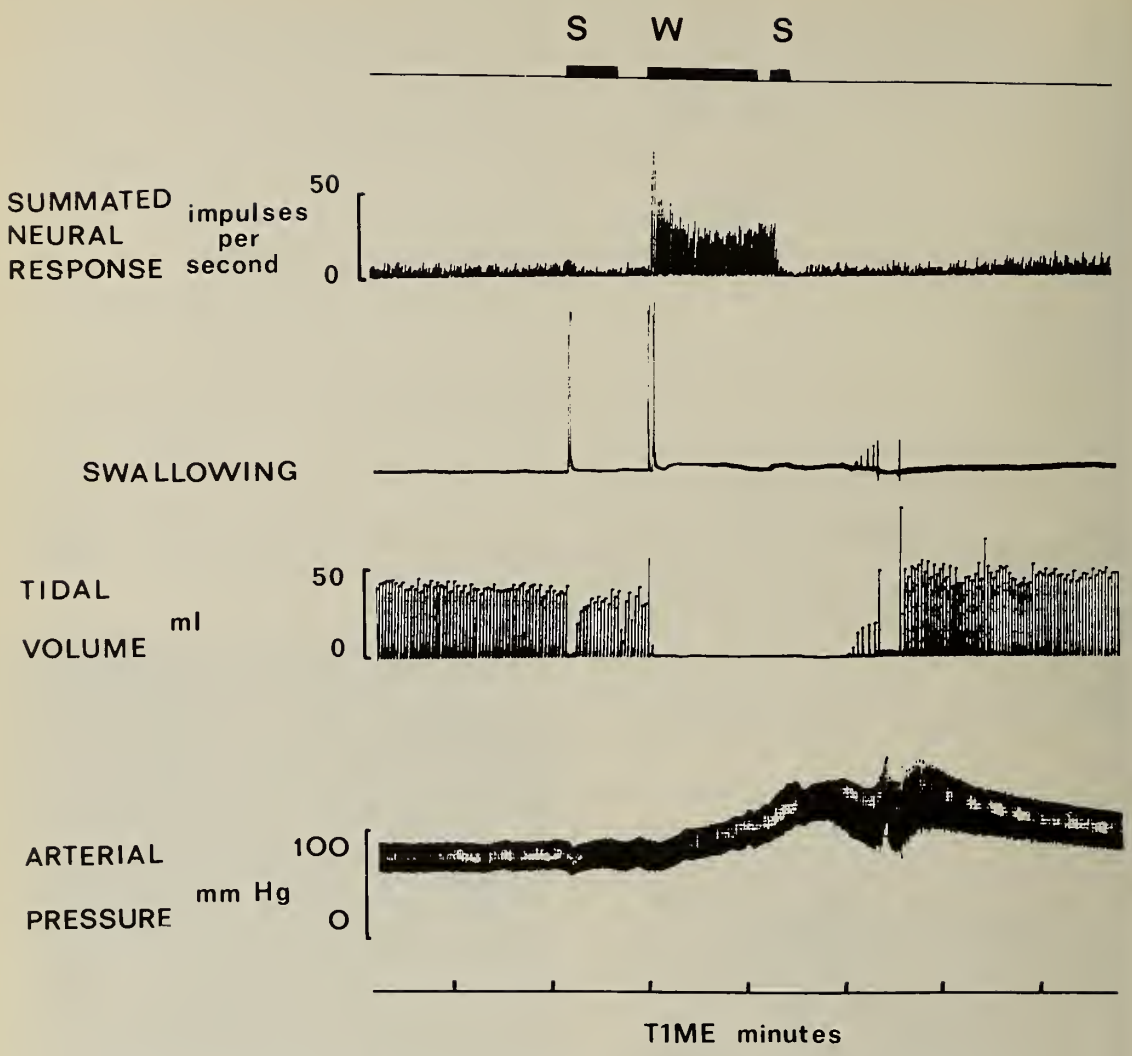


Figure 8–6. Effect on superior laryngeal nerve (SLN) summated response and tidal volume swallowed of successive perfusion of upper airway with Saline (S), Water (W) and Saline (S).

Water introduced into the larynx evoked a continuous discharge so long as the water stimulus was present. This activity was promptly suppressed when saline replaced water as the stimulus. Figure 8–6 shows a typical summated single unit response. R. Harding has recorded responses of single SLN units to cow's milk, water and various concentrations of sodium chloride. It appears almost as if the SLN provides a "bio-assay" of NaCl. As the concentration of saline is decreased, both the frequency of single unit discharge and the degree of apnoea increase. A population of such units could be responsible for the reflex respiratory events. The developmental change from persistent to transient apnoea may well relate to a central reorganization of these as well as other sensory inputs.

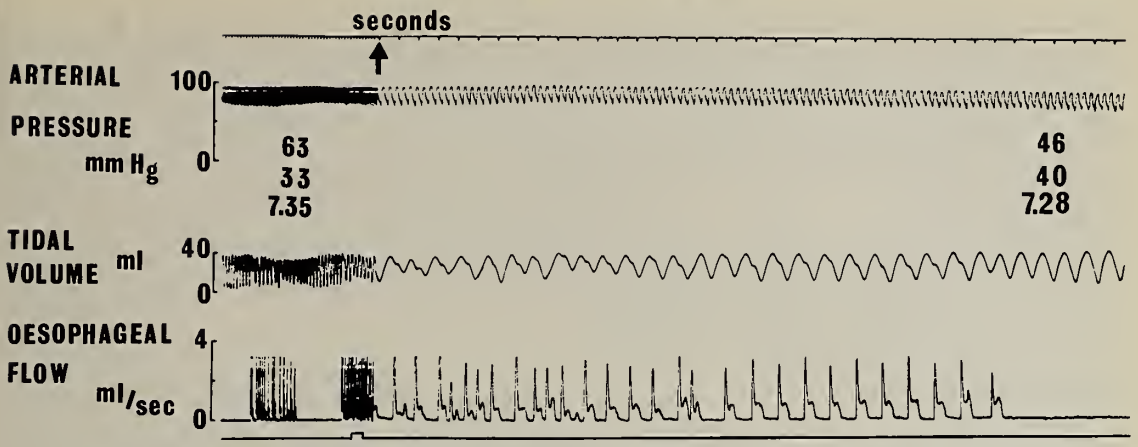


Figure 8–7. Recording of blood pressure, respiration and esophageal flow from a lamb during suckle feeding of ewe's milk. At arrow, paper speed was increased × 8. Numbers on the chart are Pa 0_2, Pa $C0_2$ and pH, respectively. See text for description of methods and interpretations.

Feeding and state. In order to study the functional significance of these responses we adopted the more realistic approach of introducing the fluids from the oral direction. Presumably this is the more commonly used pathway. Figure 8–7 is a record from an unanaesthetised newborn lamb with a flowmeter in the oesophagus for measuring swallowing. A pneumotachograph provided quantitative measures of ventilation and an arterial catheter measures of blood pressure. The lamb ingests ewe's milk from a bottle. This animal is quite capable of continuing to breathe, seemingly fairly effectively, during spontaneous feeding. However, it is worth observing that at the end of a relatively short swallowing burst this lamb was comparatively hypoxemic and hypercapnic. We were impressed that these animals were apparently more "interested" in food intake than the associated asphyxia. We have even observed lambs that are standing while feeding to develop a PaO_2 as low as 20 torr (normal PaO_2 approx. 80 torr.). It appears that feeding represents at least one physiological situation in which the chemical drive to respiration is suppressed and effective breathing does not occur. Attention must be focused on effective breathing and not merely on breathing *per se.*

By gently inserting a soft oropharyngeal catheter in newborn lambs we were able to introduce fluids directly into the pharynx. Breathing, swallowing and heart rate were monitored non-invasively while saline or water was placed in the pharynx during sleep. Water usually initiated a startle, swallowing and transient apnoea. Occasionally we observed apnoea alone. In such cases the apnoea was likely to be prolonged and persistent and to terminate in a convulsive episode. On the other hand, saline would usually not initiate swallowing or apnoea and would be aspirated into the lower airway. Often explosive coughing, presumably initiated from the carina would both wake the lamb and remove the offending liquid. It was noticeable that the lamb did not have a cough reflex initiated from the laryngeal aditus either by

direct stimulation or electrical stimulation of SLN. These studies did not include a detailed analysis of sleep state which may have provided a better guide to the pattern of response observed.

These various studies suggest the possibility that swallowing, breathing and cardiovascular responses of the human infant during feeding might depend upon the nature of the liquids presented. It is likely that the magnitude of the responses and thus the ability to measure differences between specific liquids depends on involving the chemosensory defense of the airway.

HUMAN STUDIES

The previous observations in animals suggest that the nature of the nutrients fed determines not only the feeding pattern but also breathing pattern. It was felt that one could reasonably study the human infant and ask some of these questions in a physiological and ethical manner. Thus some normal, healthy, alert term infants were fed saline and water.

Methods

To obtain accurate measurements, rather than for any physiological consideration, these infants were fed from a special bottle that prevented the development of a vacuum during sucking. Thus a constant *ad libitum* supply of fluid was available to the infant at low resistance. The clear perspex bottle, which is pictured in Figure 8–8, incorporates an electromagnetic flowmeter in its outlet and a reed valve that prevents the development of a vacuum inside. The flow signal can be integrated to provide net flow per unit time when required. Breathing was recorded by measuring tidal CO_2 via a nasal catheter to a Beckman LB1 analyser. Heart rate was usually measured from skin ECG electrodes. In some infants, laryngeal movements were measured from pressure changes in an air filled balloon adherent to the skin of the neck above the larynx. The electrical signals were displayed on an oscilloscope and recorded on a 4 channel UV/physiological recorder.

It was necessary for the electromagnetic flowmeter to remain liquid filled in order to function accurately. As a byproduct of using this system the amount of excess air swallowed was reduced and the quality of the sucking records improved. Neither flowmeters nor infants function particularly well when excessive amounts of air enter them.

Saline and water

Term newborn infants delivered in the John Radcliffe Hospital and the products of normal pregnancies were randomly selected. They were studied during the first week of life at routine feeding times after they had settled down with the monitors applied. In the initial studies the infants were fed 30 milliliters (mls) of isotonic saline and water according to a selected se-

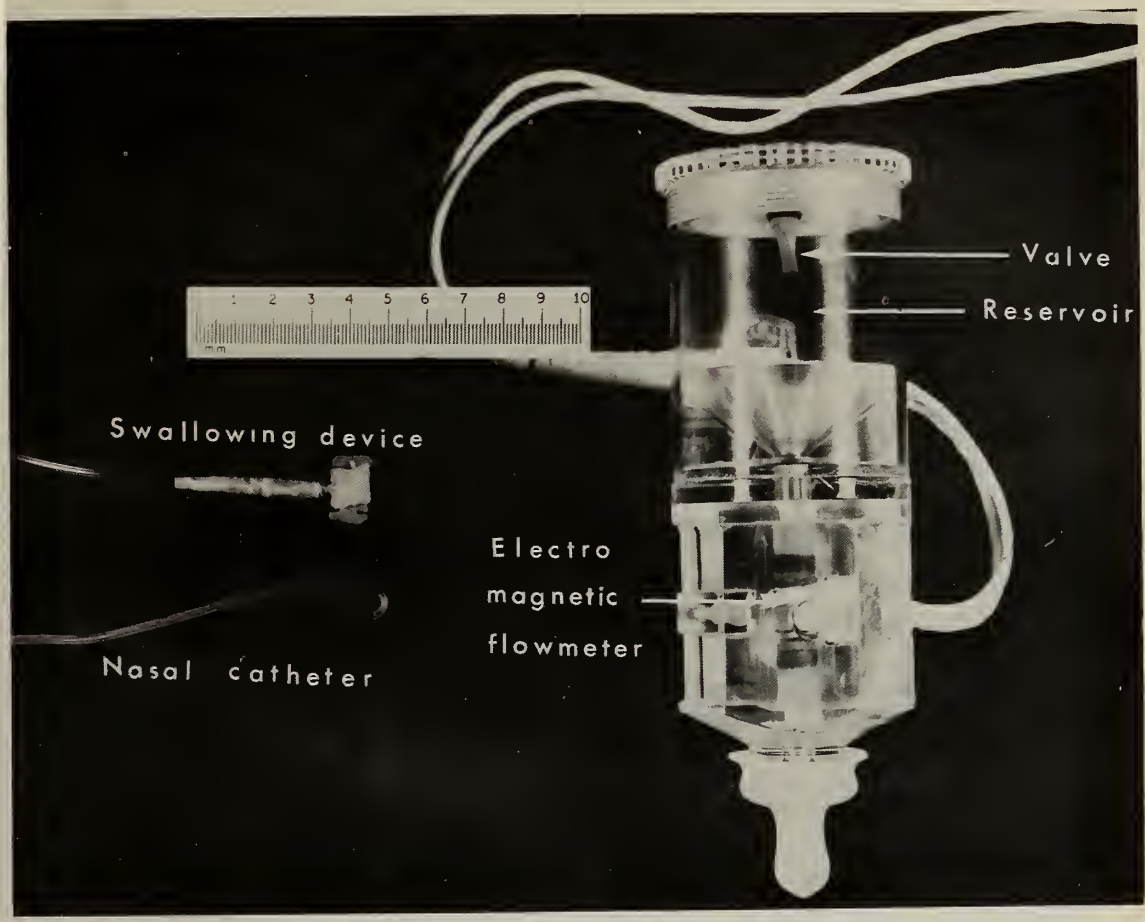


Figure 8–8. Nursing apparatus with electromagnetic flowmeter and controlled valve to atmosphere.

quence. During feeding each infant was held by a nurse assistant or by the mother.

An interesting and perhaps alarming finding is seen in Table 8–II. While swallowing the saline, 7 of the 10 newborns aspirated an appreciable quantity of it. Similar gurgling in the airway, however, was detected in only 1 of 8 infants who were over one week of age. None of the babies came to any harm, but these particular studies ceased. The results suggest that the control of sucking, breathing and swallowing in the human might involve sensors and reflexes similar to those that we had been examining in the lamb. The next studies examine responses to the liquids that human babies would normally receive, artificial formula and human breast milk.

TABLE 8–II

INHALATION OF FLUID BY HUMAN INFANTS FED SALINE

Age	*Total sample*	*Inhaled saline*
1–2 days	10	7
6–18 days	8	1

Breast and artificial milk

Forty-four newborns were fed expressed breast milk (EBM) and artifi-

cial formula;* each for one minute. The EBM was from a breast milk bank and not from the individual infant's mother. The sequence in which the fluids were given varied. Infants drank approximately 30 ml. of each fluid. The behavior of the infants in response to EBM provided a division of the sample into two groups of approximately equal size. The infants of Group 1 showed a characteristic burst-pause pattern of sucking like that shown in Figure 8–9. This sucking is similar to that described by Lipsitt in this volume.[10] The infants swallow 30 ml. of EBM with virtually no disturbance of their pattern of breathing. However, on feeding artificial milk, these infants displayed the quite different pattern shown in Figure 8–10. Sucking was generally continuous over the feed period. Breathing was disturbed, often markedly so. The infants of Group 2 exhibited the disturbed pattern of feeding and breathing in response to both EBM and artificial milk. The two groups were not distinguished by any feature of the pregnancy, delivery or neonatal management we could discover. Interestingly neither the nursing staff or the mothers noticed any difference between the babies of these two groups, whether they were being artificially fed or not. Other details of this study appear elsewhere.[8]

The burst-pause pattern of sucking which we have observed is considered by Wolff [14] to be uniquely human. It has recently been described in the context of breast feeding.[12] Our observations of breast feeding made in connection with present studies are confirmatory. Furthermore, it is of interest that several mothers observing the sucking pattern on an oscillo-

Figure 8–9. Record of fluid flow, of suckling per laryngeal elevation, and of expired CO_2 during drinking of human expressed breast milk (EBM). Female infant age 4 days; b. wt. 3799g.

*This formula, baby milk 1 (half cream), is manufactured by Cow and Gate Baby Food, Guildford, Surrey, England. It is intended for use with normal newborns and contains 2.0 gm. fat, 2.5 gm. protein, 7.1 gm. CHO, 55 calories per 100 ml.

Figure 8–10. Record of fluid flow, of suckling and of expired CO_2 during drinking of artificial formula (see footnote page 120). (Same infant as Figure 8–9)

scope commented that the burst-pause pattern was similar to their experience of breast feeding their infant. The record in Figure 8–11 confirms the point made by Lipsitt that many babies sucking from an empty bottle or a blind teat exhibit the burst-pause pattern and demonstrate the restricted extent of breathing disturbance.

In a number of instances, bradycardia was seen as part of the response pattern shown in Figure 8–10. We believe this may be a primary reflex response via chemo-sensitive receptors or a secondary effect of the relative hypoxemia during the hypoventilation. These factors cannot be assessed in the human as yet. However, evidence suggesting differential cardiorespiratory response to artificial and breast milk comes from two term babies that became cyanotic on feeding artificial milk. Under our observation these infants did not become cyanotic when

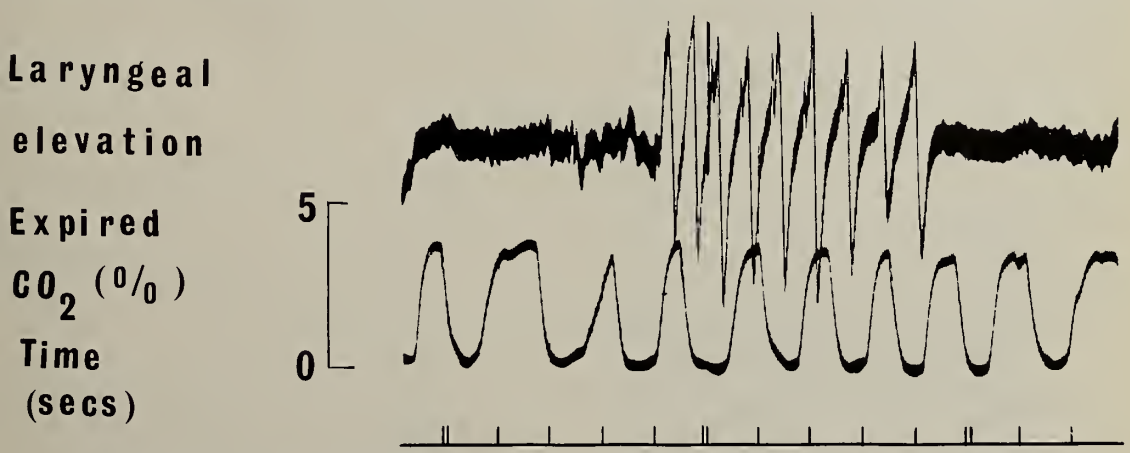


Figure 8–11. Breathing as measured by expired CO_2 continues in regular pattern during non-nutritive sucking.

fed either EBM or saline. Cyanosis during feeding is not an unusual clinical problem. A large delivery service can be expected to have 2 or 3 such infants per year. They appear to outgrow the cyanotic episodes in response to feeding. Similar observations have been made by E. Bernard in Sidney (unpublished observations).

CONCLUSIONS

It seems, therefore, that the chemo-sensitivity of the upper airway influences feeding and breathing patterns. Whether humans have functional sensors at the larynx and a reflex similar to the one we have observed in lambs, we cannot say. However, the fact that some babies seemingly fail to close their airway when feeding saline, does suggest that such a mechanism is involved during normal feeding. The clinical significance of these differences remain to be elucidated.

At present, it seems that taste or something very like it is an important component of the reflex defense of the airway during suckling. When testing the effects of various liquids on performance during ingestion, one should be aware of possible simultaneous effects on ventilation. Feeding is a complex integrated reflex function with respiratory and heart rate components that fortunately can quite readily be measured non-invasively. At the very least, one should be aware of the hypoxaemia that may develop during feeding. It is known that hypoxaemia, particularly in the newborn, can depress a number of functions including breathing and swallowing.

Our observations in the human were carried out on infants in the alert awake state. This may be critically important. We are presently studying changes in the functional response of neural pathways involved in the defense of the airways in relation to the normal rest-activity cycle and diurnal rhythms in the newborn. In particular, we are considering this type of reflex response in relation to apnoea of the premature human newborn, and its possible role in the sudden infant death syndrome.

REFERENCES

1. Boddy, K., and Dawes, G.S.: Fetal breathing. *British Med. Bull.*, 31:1, 3–7, 1975.
2. Boddy, K., Dawes, G.S., Fisher, R., Printer, S., and Robinson, J.S.: Fetal respiratory movements, electrocortical and cardiovascular responses to hypoxaemia and hypercapnia in sheep. *J. Physiol.*, 243: 599–618, 1974.
3. Bosma, J.F. Discussion. In Bosma, J.F. (Ed.) *Fourth Symposium on Oral Sensation and Perception*. Washington, D.C., Government Printing Office, 1973, p. 306.
4. Bradley, R.M., and Mistretta, C.M.: The gustatory sense in fetal sheep during the last third of gestation. *J. Physiol.*, 231:271–282, 1973.
5. Dawes, G.S., Fox, H.E., Leduc, B.M., Liggins, G.C. and Richards, R.T.: Respiratory movements and paradoxical sleep in the fetal lamb. *J. Physiol.*, 210:47p–84p, 1970.

6. Johnson, P.: Laryngeal induced apnoea. In Robinson, R. (Ed.) *Sudden Infant Death, 1974.* Toronto, Canadian Foundation for the Study of Infant Deaths, 1974.
7. Johnson, P., Robinson, J.S. and Salisbury, D.S.: The onset and control of breathing after birth. In Comline, K.S., Cross, K.W., Dawes, D.S. and Nathanielsz, P.W. (Eds) *Barcroft Symposium on Fetal and Neonatal Physiology,* Cambridge, Cambridge Univ. Press, 1972, pp. 217–221.
8. Johnson, P., and Salisbury, D.M.: Breathing and sucking during feeding in the newborn in maternal infant relationships. In Porter, R. and O'Connor, M. (Eds.) *Parent Infant Interaction,* Amsterdam, Elsevier Excepta Medica (in press).
9. Lalonde, E.R., and Eglitis, J.A.: Number and distribution of taste buds on the epiglottis, pharynx, larynx, soft palate and uvula in a human newborn. *Anat. Rec.,* 140:91–95, 1961.
10. Lipsitt, L.P.: Taste in human neonates: Its effects on sucking and heart rate. (this volume)
11. Mistretta, C.M., and Bradley, R.M.: Taste *in utero*: Theoretical considerations. (this volume)
12. Luther, E.C., Arballo, J.C., Salan, L. and Cordero Funes, J.C.: Suckling pressure in humans: relationship to oxytocin-reproducing reflex milk ejection. *J. Appl. Physiol.,* 36: 350–353, 1974.
13. Storey, A.T., and Johnson, P.: Laryngeal water receptors initiating apnoea in the lamb. *Exp. Neurology,* 47:42–55, 1975.
14. Wolff, P.H.: Sucking patterns in infant mammals. *Brain Behav. Evol.,* 1:354–367, 1968.

DISCUSSION

MISTRETTA: In regard to the blood gas values which you have measured in feeding lambs, have you ever observed lambs that continued to feed even though blood gas values are dangerously abnormal?

JOHNSON: Usually these animals had exhausted the milk in the bottle at the same time as they were observed to be hypoxaemic. However we have a number of observations of lambs standing still feeding with PaO_2 between 20 and 34 (normal value newborn lamb approx. 80 torr.). Most of us would have considered these levels to be significant hypoxaemia in terms of the survival of these lambs. It does become difficult to say just how dangerous the hypoxaemia, presumably repetitive, may be. As far as the human is concerned, in the two infants that were cyanotic during artificial feeding but not when fed breast milk, the heart rate changes and the visible appearance of cyanosis with time, give us some indication. We hope to obtain a transcutaneous oxygen and CO_2 electrode for the human studies to obtain a more definitive relationship of the blood gas status during feeding. We, also, plan to come back to this in more detail in animal studies.

PFAFFMANN: I would like to relate your records from the very posterior oral cavity to the question of tongue responsivity in the lamb. First, what about water responses?

MISTRETTA: No, water responses were not present on the tongue of fetal, newborn, or adult sheep.

PFAFFMANN: But Dr. Johnson has evidence for its occurrence at the very posterior region. That would make a lot of functional sense. Zotterman [1]

[1] Zotterman, Y.: The response of the frog's taste fibers to the application of pure water. *Acta physiol. Scand.,* 18:181–189, 1950.

theorized that the lingual water receptors protected frogs from swallowing too much water. A water receptor in the mammal makes functional sense in the control of respiration.

JOHNSON: What do you think? Can taste buds detect water? Or are these free nerve endings?

PFAFFMANN: Well, until you reported this sort of observation, one could account for a lot of the reported water responses from the tongue as a result of adaptation by other chemicals that seemed to change the excitability or the ion distribution around those taste buds. Some (Beidler [1]) doubt that the water receptor on the tongue is a true phenomena. I think Linda Bartoshuk [3] especially is concerned about that and her evidence suggests that it is a question of taste bud adaptation.

MISTRETTA: I think that taste receptors in the oral cavity located in extralingual areas could be functionally quite different from those on the tongue. Neurophysiologists should begin to study these extra-tongue taste buds in more detail.

NOWLIS: Perhaps Dr. Johnson's data lend themselves as well to an interpretation involving taste bud adaptation. If the fluids in which the taste buds in this study are bathed have a sodium ion content close to that of physiological saline, then we might infer that this is the concentration of sodium ion at which the taste buds are adapted. Further stimulation with applied physiological saline would not be expected to yield a taste response, whereas lower concentrations or distilled water would do so, according to the current views on the water response cited by Dr. Pfaffmann. Dr. Johnson's report of a reflexive response to water but none to physiological saline would seem consistent with this view.

JOHNSON: It is not necessary for the water to follow saline, if you're referring to our experiment. We are now examining the effects of saliva and regurgitated gastric contents and this sort of thing that may, very reasonably, come in contact with this area and modify these responses.

[1] Beidler, L.M.: Anion influences on taste receptor response. In Hayaski, T. (Ed.), *Olfaction and Taste, II,* Oxford, Pergamon Press, 1967, pp. 509–534.

[2] Bartoshuk, L.M.: NaCl thresholds in man: Thresholds for water taste or NaCl taste. *J. comp. physiol. Psychol.,* 87:310–325, 1974.

Chapter 9

TASTE IN HUMAN NEONATES: ITS EFFECTS ON SUCKING AND HEART RATE

Lewis P. Lipsitt

The pacifying effects of sucking on agitation in the human newborn have been appreciated for many, many years. Tradition in lying in hospitals has it that a sugar nipple, which is a commercial bottle nipple stuffed with a wad of cotton soaked in simple syrup, works even better. The sugar nipple is often used in the course of otherwise unanesthetized circumcisions to reduce the wailing, and presumably to increase the comfort of the infant.

The first section of this paper will present a brief historical summary of data on neonatal taste existing to about 20 years ago, at which time a greater empirical interest began to appear in all aspects of sensory functioning in the very young child. Not surprisingly, the upsurge of interest in perceptual processes of the newborn and in the hedonic (reinforcement-guided) quality of his responses has coincided with a contemporary boom in research on learning capacities of the very young child.

The second portion of the paper will present some recent data from my own laboratory relating to the effect of taste on several parameters of the sucking response, the stability of sucking behavior within the newborn period, and some findings concerning the relations among taste, sucking, and heart rate. In this section it will be shown that increasing sweetness of the fluid delivered to the infant contingent upon its sucking response results both in a slowing down of the sucking rate *and* an enhancement of heart rate, suggesting that a savory (positively hedonic) influence is at work. It will be obvious that more research is needed on the integration of sucking, breathing, swallowing, and heart rate, and the role that early taste and feeding experience may have in determining later appetitive and aversive behaviors.

NEONATES TASTE SWEET

What follows is a chronological summary of the early literature on neonatal reactions to gustatory stimuli.* The dearth of such studies, coupled with the lack of standardized apparatus, stimuli, and methods has slowed the acquisition of knowledge about the taste sense in infants. Moreover: "The absence of language responses in the newborn infant has restricted the investigation of just

* This review was prepared with the help of Sally Ann Serunian, to whom the author is grateful, and its preparation was supported by funds from the National Institutes of Health.

those problems which are the basis of adult behavior. If some substitute for such responses could be secured, a new field of inquiry would be opened and light shed on many problems which had not even been formulated" (Jensen, 1932, p. 367). Forty-three years later, it still must be regretted that no method has yet been developed which yields unequivocal conclusions pertaining to the positive and negative taste reactions of neonates.

Because the newborn's gustatory responses are not entirely like those of adults, some early investigators were led to believe that the taste sense in neonates is "dormant," [1,28] while the fact that some differential reactions are found at birth led others to conclude that the taste mechanisms are already well developed at that time.[15,23,27]

Most of the early psychobiological research on taste in infants has been extensively reviewed by Peiper,[22] and Pratt, Nelson, and Sun.[26] Pratt (1954, pp. 242–244) has also provided a brief synopsis. In the earliest reported work, Kussmaul [15] used a saturated sugar solution, and solutions of quinine sulphate, salt, and tartaric acid as stimuli. Facial expression and movements of specific parts of the face were the responses observed. Kussmaul noted sucking movements to sugar, and grimaces to the other three stimuli. Occasionally, however, he found the facial expressions to sugar similar to the reactions to bitter. He also found that even infants born prematurely by one or two months would suck at sweet substances with what appeared to him to be satisfaction, whereas ". . . upon the application of quinine solution the face became distorted, the tongue would be stuck out, and the introduced liquid ejected with abundant spittle" (Kussmaul, quoted in Montagu, 1962, p. 106). On the basis of these results, Kussmaul concluded that the taste mechanism in newborn infants is well developed and that gustatory stimuli, for the most part, produce facial expressions corresponding with those of the adult.

Preyer [27] seemed to substantiate Kussmaul's findings, again using facial expressions. His stimuli were sugar, salt, an acetic acid solution for the sour stimulus, and quinine (3–5% solution) for bitter. Preyer's descriptions indicate that quinine usually evoked a wry facial reaction, characterized by tightly shut eyes, convulsive contractions of the throat, a wide opening of the mouth, and the ejection of the solution along with mucus. Even on the first day of life, this "bitter" expression was reportedly quite different from the one to "sweet." In turn the "sour" expression differed from both the "bitter" and the "sweet" reactions. Preyer observed individual differences in reactions of the newborns to the various taste stimuli, differences which he explained in terms of "sensibility to degrees of intensity" (Pratt, Nelson, and Sun,[26] p. 105). He suggested that neonatal sensitivity can not only be measured to differing intensities of taste stimulation but that sensitivity increases in the first few days of life. He concluded that of all reactions in the newborn, those to gustatory stimuli are most highly developed and that differential gustatory qualities are distinguished by the neonate, evoking different reflex movements. Preyer's conclusions were essentially

the same as those of Kroner,[14] writing at about the same time.

Markedly contrasting with the observations of specific reactions to taste stimuli, Shinn [28] reports that the newborn is extremely unresponsive to different tastes on the basis of her repetitive observations of one child.

The work of Peterson and Rainey [23] is noteworthy for the large number of subjects observed. They observed the facial reactions of 76 neonates to a concentrated salt solution, a 1% solution of acetic acid, syrup of unspecified strength, and tincture of gentian. They reported differential mimetic reactions to these four stimuli characteristic of adults. Besides these expressions of contentment or grimaces of discomfort, they found that the simple appearance of sucking movements cannot be taken as evidence of satisfaction, for salt, sour, and bitter stimuli often produce such sucking motions conjoined with expressions of dissatisfaction. Peterson and Rainey found the same reactions in prematures as in term infants.

Canestrini [3] seems to have been the first investigator to use direct measures of cardiovascular and respiratory response for studying neonatal taste reactions. He attached a Mare pneumograph to the top of the infant's head and recorded the infant's pulse from the fontanelle. A small pneumobelt around the infant's abdomen recorded respiration. Canestrini studied the responses of 35 subjects from one to fourteen days of age to the following solutions, warmed to body temperature: (a) sweet (2–5% sugar solution), (b) sour (2–5% vinegar solution), (c) salt (2% solution), (d) bitter (2% quinine solution), (e) diluted cow's milk, (f) mother's milk, and (g) air. A quieting, calming effect of the sweet solution was shown by a lowering of the respiratory and circulatory rates. Salt produced "restlessness," revealed in respiration disruption and pulse increases, and by cessation or interruption of sucking movements. Sour and bitter produced irregularities of breathing and fontanelle action that were greater than those caused by sweet and salt. Responses to sour and bitter stimuli were not grossly different from each other.

Canestrini reported that response latency to taste stimuli was shorter than for any other neonatal sense mechanism. This contention, while impossible to support definitively because of the dependency of response latency on stimulus intensity and the difficulty of making cross-modal comparisons, finds some corroboration in modern observations. The newborn's sucking rate, as we now know, changes within just a few sucks after a shift from one concentration of sugar to another.[18] Canestrini's observation that with every sucking movement there is an increase in fontanelle action, while probably incorrect quantitatively except under specially selected taste conditions, indicates that he was sensitive to the intricate interrelationships among the several psychobiological systems associated with taste and ingestion (sucking, swallowing, respiration, and pulse) whose synchrony we are now beginning to understand quite well. Canestrini also noted that the infant's high degree of gustatory sensitivity has survival value, in that it operates to protect the young child from ingesting harmful solutions.

Drummond [5] wrote of the difficulty

involved in isolating taste from smell, temperature, and touch. Using the infant's behavior and facial expressions as indicators of satisfaction or dissatisfaction, she concluded that any moderate taste stimulus is pleasant, and a very strong stimulus is unpleasant and gives rise to grimaces and movements calculated to avoid it. Drummond was, then, cautioning against contaminating stimulus quality with stimulus intensity.

Eckstein [7] criticized mimetic responses as crude indices of differential taste sensations, and his studies of prematures, normal neonates, and older infants used graphically recorded sucking responses. The stimuli were cane sugar, salt, quinine, and lemon juice, all in varying concentrations. Eckstein's results showed that even premature infants respond differentially to different taste stimuli. Almost all infants made similar differential responses to the same stimuli, with "bitter" almost without exception being followed by inhibition of the sucking reflex.

Another study worthy of brief mention was that of Bryan [2] who tested 66 neonates with a variety of stimuli in different modalities and found no differential reactions to various milks which were presented as taste stimuli, this finding being in agreement with that of Canestrini, who found no differential response to cow's milk and mother's milk. Unfortunately the report does not provide much information on the types of observations made.

The Pratt, Nelson, and Sun [26] investigation of neonatal taste, like their studies of newborns in other modalities, stands as a classic because of the parametric administration of stimuli and the care taken in quantifying babies' responses. They used a stabilimeter and polygraph to record bodily movements, and obtained supplementary records of sucking behavior and facial reactions. The infant's mouth was opened for insertion of the taste applicators (cotton-tip sticks) by touching the corner of the mouth. In fact, the authors noted that this tactile stimulation was sometimes sufficient to induce sucking, and in their response recording they distinguished between the sucking reflex and the infant's reactions to taste stimuli. Testing 28 infants an average of 8 times from birth to 15 days of age, for a total of 227 tests and 858 stimulations, Pratt, Nelson, and Sun observed differential responses to sugar, salt, quinine, and citric acid, with distilled water being used as a control stimulus. Temperatures of the solutions were kept relatively homogeneous between 20 and 27 degrees Centigrade.

The sugar solution used was 5 grams to 30 grams of water (16.66%), the salt solution was 5 grams to 60 grams of water (8.33%), the quinine was .3 grams to 120 grams of water (.25%), and the citric acid was 3 grams to 140 grams water (2.14%).

Pratt, Nelson, and Sun presented tables to show that they obtained some reaction to all of the non-water stimuli about 85% of the time, with sucking occurring on approximately 30% of the stimulations. There was relatively little change in frequency of elicited sucking from birth to 11 days of age, but it was noted that head movements increased from 7 to 11%, and facial reactions increased from 14 to 24%. Facial reactions became particularly pronounced, the authors said, to salt,

sour, and bitter. Over the first 11 days of life, sucking movements for sugar increased from 44 to 52%, and water 26 to 37%, and decreased for quinine (20 to 16%) and citric acid (6 to 4%). Overall sucking reactions to the various stimuli were: 49% to sugar, 36% to salt, 32% to water, 22% to quinine, and 7% to citric acid. The reactions to water seemed to suggest that newborns react not to taste alone but in part to the presence of something in the mouth, and thus response to water should perhaps be taken as the "basal level." It was found that body movements occurred most (25% of the time) to citric acid and least (15%) to water. Interestingly, Pratt, Nelson, and Sun compared adult reactions to those of the infants and observed that the citric acid solution which they used seemed weak to adults, yet produced strong reactions in the infants; conversely, their quinine solution seemed strong to the adults, but produced a relatively weak aversive response in the neonates.

In another early investigation with well-controlled procedures for the time, Jensen [10] used a simulated feeding procedure as the setting for recording infant responsivity to taste stimuli. Rather than dabbing the tongue with minute quantities of fluid, Jensen used a specially constructed nursing bottle. Its calibrated nipple connected with a manometer that registered responses on a kymograph. The taste stimuli were glucose (6% solution), acid milk, sterile water, and five dilutions of salt solution (.900, .450, .300, .225, and .200%). The control solution against which response to these tastes was compared was milk at 40 degrees Centigrade. The infant was first given the control solution for 10 seconds, then the experimental mixture for 10 seconds, and finally the control solution for 10 seconds. Infants were tested for about 15 minutes before and after each regular feeding period, to provide data on effects of hunger and satiation on taste.

Four distinct patterns of sucking behavior were classified by Jensen: (1) sucking occurs for a few seconds, followed by a short break after which sucking is resumed, (2) continuous sucking with varying pressures, (3) initial sucking, followed by a pause or an amplitude decrease, then resumption of sucking, followed by other manifestations of variability, and (4) found most frequently, persistent sucking for an interval of from three to twelve seconds. These four types of reactions are rated coincidentally with the administration of the experimental or control solutions, with the nipple remaining in the infant's mouth.

All fourteen infants tested failed to show a reaction to the .200% salt solution, one showed it to the .225% solution, and six to the .300% solution. On the other hand, only three failed to respond differentially to the .450% solution, and only one to the .900% solution.

When differential reactions occurred, 95% of the time they were without marked facial expressions. When a facial change did occur it was always accompanied by a differential sucking response. Jensen observed that there were large individual differences in taste thresholds, as can be noted from the proportions of subjects responding to the graded salt solutions, and that the thresholds for the same infant tended to remain constant throughout the period of experimentation. The differential

reactions secured were not of an all-or-none character, but proved to be gradual deviations from the control-period solution, being more marked when the temperatures of the experimental solutions were raised or lowered relative to the control solution.

Jensen noted that vigorous avoidance movements were made, and crying occurred, when the salt solution was less diluted. Interesting also was the observation that the moderately satiated baby was a better discriminator of taste than the very hungry infant. Only salt solutions among the gustatory stimuli, moreover, impaired or caused cessation of the sucking response. This finding has its modern counterpart in the work of Crook (See pp. 210).

Using taste applicators similar to those of Pratt, Nelson, and Sun, Shirley [29] observed facial and bodily reactions of 14 newborns to bitter, salt, sour, and sweet stimuli. She reported that reactions to the first three were mainly those of rejection, indicated by turning the head from side to side, making a wry face, crying or fussing, pushing the applicator out of the mouth with the tongue, or waving the arms with the effect of removing the applicator from the vicinity of the mouth. Twelve of the fourteen infants from one to fourteen days of age were reported to suck contentedly on the sweet applicator, with some facial reactions present.

Sonohara [30] used the recorded sucking response as a measure and found that infants responded from the first day to a saccharin solution with sucking motions. Initially, Sonohara says, the threshold is considerably higher than that of adults, but an approach to the adult threshold occurs by seven days of age. Sonohara observed that sucking to saccharin will occur when the infant is drowsy but not during sleep, an observation counter to that of most neonate researchers, who do not report failure to suck during apparent sleep.

Stirnimann [32] reported a detailed description of observations on 100 neonates (including 7 prematures). The gustatory solutions were applied to the tongue by means of a nipple-shaped cotton piece. From birth, taste reactions to sugar were observed in all subjects, and Stirnimann reported that there were reactions corresponding to the four principal taste sensations. These reactions were rather variable, however, and included sucking responses, attempts to approach or retreat, gestures, and crying. Sucking responses followed not only stimulations with sugar, but also those with salt, citric acid, and quinine. Discriminative mimetic reactions were occasionally observed. Infants were noted to react more vigorously to a 7% saccharin solution than to a 7% lactose solution, more vigorously to a 16% solution of lactose than to the weaker 7% lactose solution. No differential responses were found among .9, 1.5, and 2.0% salt solutions. Stirnimann noted that infants sometimes cry when sugar-soaked applicators are removed from their mouths.

Up to the end of this review, the results of different investigators show that no method has yet been developed making possible unequivocal statements as to the gustatory sense in neonates. It is still uncertain whether the newborn infant accurately differ-

entiates all four taste qualities. The evidence does indicate, however, that infants give a positive reaction to sweet, and that salt, sour, and bitter may give negative reactions. It appears that salt solutions tend to interrupt the sucking response, whereas sugar solutions evoke and sustain it. Acid solutions to a lesser degree elicit sucking, and quinine solutions seldom do. Facial reactions mimetic of "dissatisfaction" are most conspicuous to quinine. Generally, respiration and circulation are least influenced by "sweet," slightly disturbed by "salt," and considerably affected by "sour" and "bitter." Interpretation of data from neonatal taste experiments is complicated by the fact that almost any stimulation of the oral cavity and lips will evoke sucking behavior. It appears that a procedure for assessment of taste reactions will be most suitable if the delivery of the gustatory stimulus is through a suck-inducing nipple placed and maintained in the infant's mouth throughout the testing period. Taste and palatability might then be phrased in terms of a suppression ratio, cast as a proportion relative to the amount of sucking obtained to an optimizing taste stimulus (e.g., 10% sucrose) and an optimizing nipple (e.g., a commercial nipple approximating the mammary apparatus of the maternal delegate of homo sapiens). First we must find out what the sweet taste does, to man and child.

SWEETNESS MAKES THE HEART GO FASTER

As may be discerned from the foregoing historical presentation of infant studies of gustation, most of the empirical attention to date has been in answer to the question of whether very young infants can sense and discriminate different taste stimuli. The indicants of such sensory reception and differentiation have involved typically some aspect of sucking (mouthing) behavior, sometimes coincident with facial expressions. Even recent studies such as those of Steiner,[31] Weiffenbach and Thach,[33] Maller and Desor,[19] and Nowlis [21] capitalize largely upon oral action (tongue movement, swallowing, diverse lip responses, and so on) to document the "reception" which greets the introduction of chemicals into the oral orifice. After having been regarded so many years as a "dry behaviorist" (one who looks mostly at stimulus-response and response-reinforcement connections and allegedly forgets to honor the "whole child"), I take pleasure in reminding this group of oral symposiasts that there is more to a newborn than the mouth! I will speak about sucking behavior in relation to sweetness, but an important part of the message will be that sweetness makes the heart go faster despite the fact that, at least up to a certain point, increased sweetness has the seemingly paradoxical effect of *slowing down* the sucking behavior. The rest of my presentation will elaborate on this.

In my neonatal behavior laboratory at the Women and Infants Hospital of Rhode Island, our procedure has been to study the baby in a special testing crib housed in a sound-attenuated cubicle within the laboratory. We are presently carrying out a

series of sensory and learning studies in infants from birth to four days of age, in an effort to document the full sensory repertoire of the normal newborn child, to gain an understanding of the extent to which the baby's behavior may be modified by experiential circumstances and, eventually, to devise techniques that may yield better discriminations of the stressed or high-risk child than presently exist. Among the various conditions of testing which we utilize, an automatic nipple device enables us to obtain a polygraphic record of the baby's sucking activity, sometimes over several days and sometimes utilizing different tastants in order to observe sucking and other organismic changes which occur under conditions of differential oral stimulation. The special sucking apparatus, which was built in our laboratory,[4,13] enables a response-activated pump to deliver into the infant's mouth whichever of two different fluids we wish to make available. The automatic nipple and its ancillary equipment delivers fluid into the baby's mouth contingent upon the baby's own sucking behavior. We usually arrange, as in the studies I report today, that one drop of fluid is ejected into the baby's mouth for each suck that the baby makes. The fluid is of a constant amount (.02 ml) on each delivery. Fluid-sucking conditions can be compared with a non-fluid or dry (pacifier) sucking condition.

While we are recording the baby's sucking activity, a pneumobelt placed around the abdomen provides a simultaneous recording of respiration, and chest electrodes provide a continuous recording of the baby's heart rate. (See Figure 9–1) Signals from all of these pick-up devices are

Figure 9–1. Two-day-old infant with heart rate electrodes attached (two on chest, and the indifferent electrode on right leg), pneumobelt between the abdomen and chest, and the automatic nipple in place.

displayed on a polygraph outside the testing cubicle. The heartbeat is transformed for easier quantification by a cardiotachometer which also provides a write-out record on the polygraph. At the same time, a computer picks up the information deriving from the automatic nipple and creates a frequency distribution of inter-suck intervals (that is, sucking response rate) for each child under each condition of testing. Such measures as number of sucks per burst, number of inter-burst intervals, number of sucks per minute, mean inter-suck interval,

and so on are easily derived by the computer from the inter-suck intervals.

Figure 9–2 shows a polygraph record from a two-day-old child. The baby's sucking activity, along with respiration and heart activity are clearly seen, as is the cardiotachometer transformation. The baby is seen doing what newborns characteristically do when sucking on a nipple that, as in this case, yields no fluid. [6,34] They suck in bursts or clusters of sucks separated by pauses of several seconds duration. [11] Newborns are remarkably stable, even from day to day but especially under no-fluid sucking conditions, with respect to the number of sucks that they invest in each burst and the length of the "rests" that they take between bursts.

Although under a given condition such as when sucking for no fluid or when sucking for 10% sucrose, infants are remarkably stable with respect to these sucking parameters, nonetheless the number of sucks that infants invest in a burst is influenced enormously by the sweetness of the fluid. The sweeter the fluid, the

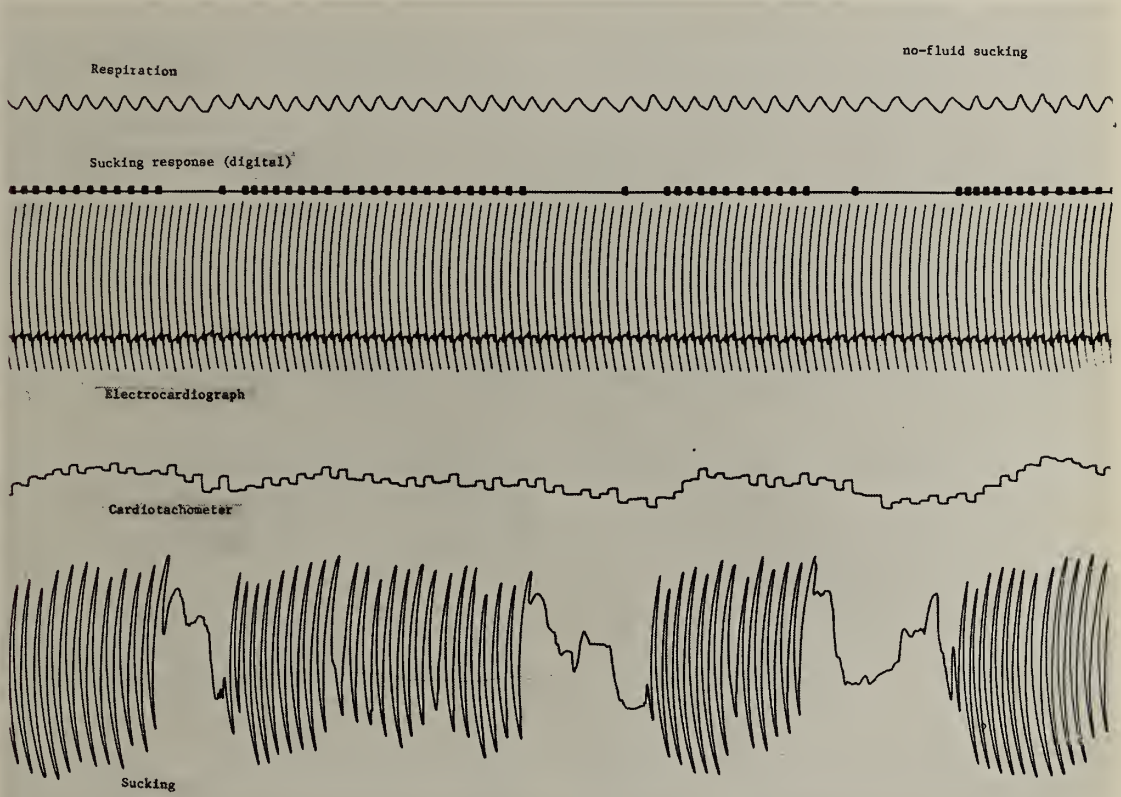


Figure 9–2. Sucking, heart rate and respiration polygraph record of a 2-day-old female during a 56-sec testing period under a no-fluid condition. The cardiotachometer provides an electronic read-out of the interbeat interval transformed into beat-by-beat heart rate. The highest heart rate during this segment is 144 bpm. Sucking occurs in bursts separated by short rests. Respiration is regular.

greater will be the length of the sucking burst. Moreover, the sweeter the fluid the shorter will be the rest intervals between those bursts. Interestingly, increased sweetness of the fluid for which the infant is sucking slows down the sucking rate within the burst even while increasing the temporal duration of the burst and the number of sucks invested in it. Quantitative expressions of these generalities will be provided shortly.

Of especial interest is an apparently paradoxical effect which we have now obtained in a variety of circumstances and over several replications. While the sucking rate decreases with increasing sweetness, the heart rate goes to and remains at a higher level for the duration of the sucking burst. The phenomenon cannot be related easily to ingestion or changes in blood-sugar levels, for the heart rate increase occurs even in the very first burst upon switching an infant from, for example, plain distilled water to 10% sucrose. Indeed, the heart rate change, like the switch in sucking rate, usually occurs within the first few sucks of the burst in which the sucrose is introduced.

It may be noted in passing, from Figure 9–2, that newborn respiration is remarkably unaffected by the sucking activity of a baby, at least under conditions of no-fluid sucking. Although our records of infants sucking for fluid delivery indicate momentary respiratory interruptions coincident with observable swallowing motions (about every four to six respiration cycles, under our small-amount conditions of fluid delivery), the respiratory cycling is remarkably resistant to disruption by sucking. It is very difficult, if not impossible, for an adult to replicate or imitate the newborn's sucking and respiration behavior in this regard.

Figure 9–3 shows a comparable polygraph record of the baby now sucking for 15% sucrose. The baby may be observed investing more sucks per burst and is now taking shorter rest periods. Despite the slower sucking rate, the infant's heart rate has climbed to a higher level than in the previous figure.

Figure 9–4 presents the data of three groups of subjects from a thesis by Kobre.[12] Groups of five subjects were compared. One group received a 15% sucrose solution contingent on its sucking behavior, another received plain distilled water, and a third obtained no fluid at all for sucking. All three groups were tested over a 20-minute session. The inter-suck intervals were cast by the computer into 100 msec bins, and frequency polygons were drawn for each group. As can be noted, there is a progressive shift of the distributions in going from no-fluid sucking to water-sucking to sucrose-sucking, such that sucking rate within bursts slows down as sweetness increases. The computer separately cast all inter-suck intervals greater than two seconds, thus defining a burst as a series of sucks all of which have inter-suck intervals less than two seconds, with the boundaries (that is, the beginning and end) of the burst demarcated by an interval of two seconds or more. The data of Figure 9–4 are sucking rates and frequencies for all sucks involving intervals of less than 2,000 msecs in the 20-minute testing period.

Another study that reflects on the relation of parameters of sucking to heart rate comes from a 24-hour test-

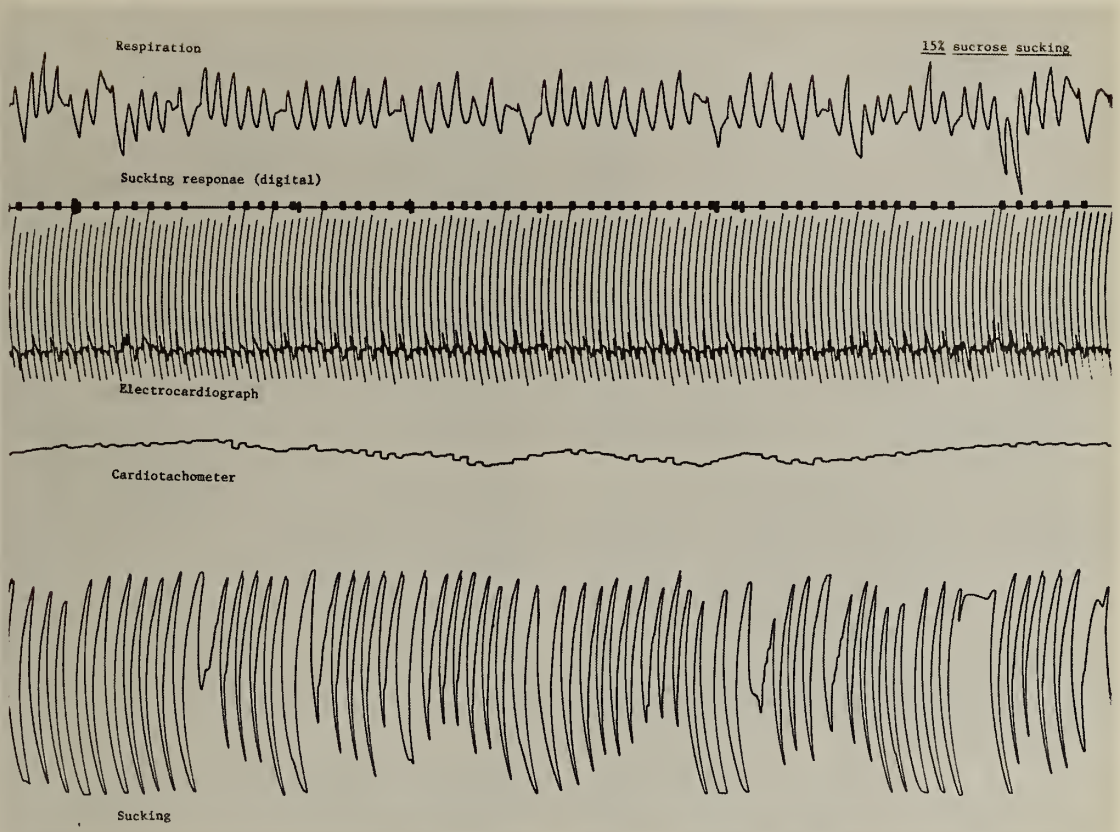


Figure 9–3. Polygraph record of same infant as in Figure 9–2, six minutes later under a 15% sucrose-sucking condition. Respiration is less regular, and heart rate higher and less variable. Highest heart rate during this period is 158 bpm, compared with 144 bpm during no-fluid sucking, even though sucking is slower. The same number of sucks (55) is nonetheless emitted during this period. Rest periods are less frequent during fluid-sucking. The larger blips superimposed on the digital sucking record are swallowing movements palpated at the neck, and coincide with observable disruptions in respiration.

retest investigation.[17] This study * is part of a larger longitudinal study exploring the possible relationships of neonatal behavioral and psychophysiological indices to later physical and psychological attributes. We first recorded the sucking behavior, respiration, and heart rate of 44 newborns, usually beginning in their second day of life. Infants were brought back to the laboratory 24 hours later for an identical regimen of stimulus administration and response recording. As part of the rather extensive behavioral testing done on each of these infants, taking approximately an hour for the complete battery, we recorded six minutes of sucking under no-fluid conditions, followed by four minutes

* The study was conducted at Women and Infants Hospital of Rhode Island. The author is indebted to the medical, nursing, and administrative staff for their cooperation over many years, and also, of course, to the mothers (and, often, the fathers) who were sufficiently convinced of the value of our studies to volunteer participation of their infants. The research has been supported by USPHS Research Grant HD 03911.

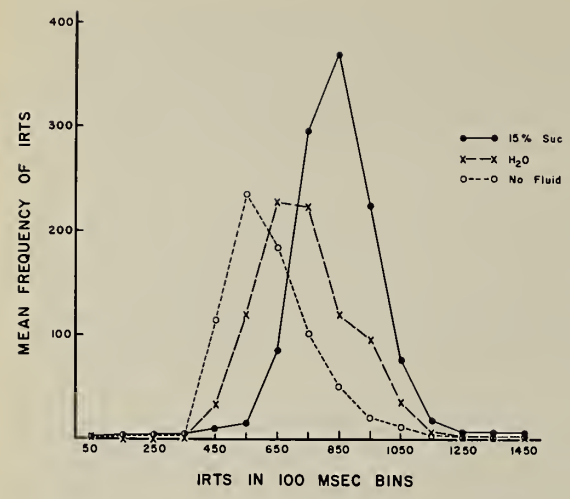


Figure 9–4. The mean number of intervals (IRTs) as a function of IRTs in 100 msec bins for the group receiving sucrose throughout, the group receiving water throughout, and that receiving no fluid at all. Each curve represents the combined IRT distribution for five subjects in a group over the twenty minute session.

of sucking under a 15% sucrose sucking condition. This enabled us to compare each baby's resting heart rate with its heart rate during nonnutritive sucking, and then during sucrose-sucking. Five measures of sucking were derived for each infant on each day to assess the day-to-day stability of the various sucking parameters. The stability of measures of respiratory and heart rates was assessed in a similar manner.

Table 9–I presents the five sucking-parameter means and standard deviations, for both days, under the no-fluid and the 15% sucrose conditions, along with the Day 1 to Day 2 correlation coefficients and probability levels for each of the parameters. For example, it may be noted that under no-fluid sucking conditions the typical number of responses invested per burst is approximately 11 to 13, while the comparable figures for that parameter under 15% sucrose sucking conditions are about 24 to 30. Similarly both modal and mean interresponse times (IRTs) are shorter (that is, rates are faster) under the no-fluid

TABLE 9–I

COMPARISON OF THE MEANS (2-MIN UNITS) OF FIVE SUCKING PARAMETERS UNDER NO-FLUID AND SUCROSE-SUCKING CONDITIONS OVER A TWO-DAY PERIOD

	Day 1		*Day 2*		*Day 1 /Day 2*	
Parameter	*Mean*	*SD*	*Mean*	*SD*	*r*	p^r
			No-fluid sucking (N=41)			
Total responses	88.3	23.2	96.6	30.9	.62	.001
IRTs > 2" (rests)	8.1	2.6	8.7	2.8	.65	.001
Responses/burst	12.9	15.5	11.2	6.1	.79	.001
Mode IRT	620.3	99.0	572.8	61.9	.37	.05
Mean IRT	657.8	81.7	611.9	56.0	.42	.01
			15% sucrose sucking (N=40)			
Total responses	103.0	28.2	106.2	27.6	.08	...
IRTs > 2" (rests)	5.3	2.7	5.9	3.5	.13	...
Responses/burst	29.7	34.2	24.2	21.7	.03	...
Mode IRT	838.1	109.9	771.3	115.7	.63	.001
Mean IRT	855.8	105.3	786.9	85.8	.71	.001

than under the 15% sucrose sucking condition, replicating the IRT data previously presented.

Next we may turn to the heart rate data. Table 9–II shows the infants' heart rates during a no-sucking rest period in relation to heart rates during no-fluid sucking and sucrose-sucking on each day. There is an orderly increase in heart rate from rest to pacifier-sucking to sucrose-sucking, with a remarkably high rate during sucking for sucrose. The correlation coefficients relating Day 1 to Day 2 heart rates show that infantile heart rate constitutes a stable individual difference variable, but most particularly under conditions where the infants are sucking for sucrose ($r=.71$, $p<.001$). The orderly heart rate increase is a significant effect.[17] Sweetness makes the heart go faster.

I should like to refer back for a moment to Table 9–I which shows the correlation coefficients for Day 1 with Day 2 measures under no-fluid sucking and sucrose-sucking conditions. It may be noted there that the most stable response under non-nutritive conditions is that involving the number of responses invested per burst, with $r=.79$. That particular response happens to be the *least* stable under the sucrose-sucking condition. The inversion of correlation magnitude from one condition to the other is essentially perfect. The most stable day-to-day measures under sucrose-sucking are the least stable under no-fluid sucking. Thus, if one wants to fully investigate the stability of individual differences in newborn sucking, it seems that one ought to test the baby under both non-nutritive and at least one sucrose-sucking condition. To indulge in a bit of theoretical reverie, perhaps it is not far from correct to assume that the responses per burst measure is a high-reliability measure of the congenital, tactually stimulated sucking response. That particular response parameter, however, becomes displaced in importance when taste experience is introduced, i.e., upon introduction of a sucrose solution into the mouth contingent upon that sucking behavior. Here the measure of sucking *rate* within bursts, indexed by the mean and modal IRT, displaces the *number of responses* measure in importance and stability. Sucking rate goes down to a low but *stable* level, and heart rate goes up very high from about 124 bpm to 146 bpm, a

TABLE 9–II

BASAL HEART RATE IN BEATS PER MINUTE COMPARED WITH HEART RATE WHEN SUCKING EITHER FOR NO FLUID OR SUCROSE AND CORRELATION COEFFICIENTS RELATING DAY 1 AND DAY 2 RATES

	Day 1		*Day 2*		*Day 1/Day 2*		*Combined*	
	Mean	*SD*	*Mean*	*SD*	*r*	*p*	*Mean*	*SD*
During rest (N = 44)	113.8	16.7	118.7	18.1	.29	<.05	116.3	14.2
During no-fluid sucking (N = 40)	124.0	14.5	123.4	13.6	.46	<.01	123.8	12.2
During sucrose-sucking (N = 39)	145.7	15.5	147.4	12.3	.71	<.001	146.6	13.1

level which is also very stable (r=.71) under the sucrose-sucking condition (see Table 9-II). The temptation is to conclude anthropomorphically that when sucrose is introduced, rate slows down to enable savoring of the tastant, and the heart rate goes up to signal the joy that the infant thus derives from this "pleasure of sensation." [24]

All sensations, even of the newborn, are, of course, not pleasant. This is of special note in relation to feeding in the neonate, for as shown in the historical section of this paper, aversive tastants have been studied by numerous investigators. The human newborn is a stimulus-rejector as well as a stimulus-assenter.

An interesting aspect of the act of feeding in the newborn is that the very act of feeding, especially breast-feeding, sometimes results in the inadvertent occlusion of both respiratory passages: the mouth, because a pressure seal is generally required and almost always effected by the baby in obtaining the milk; and the nostrils, because they are there. In the natural course of feeding, infants are often fortuitously and momentarily "smothered." This is signalled by an obvious aversive reaction (more vigorous in some than others), including head waving to and fro, arm-action, facial vaso-dilation, and crying. The objection, in infants who oppose the obstruction, is strenuous and profound (Gunther [8,9]). Mothers sometimes reflect, following such feeds in which things have not gone well, that their babies do not like them. This can, of course, lead to a reciprocating anxiety or rejection-response in the mother.

While extensive consideration of this sequence of events is beyond the scope of this paper, the occurrence of such a behavior pattern, because it is associated with the mouth and with feeding, should be acknowledged, particularly since it has been presumed (by Gunther among others) that early experiences of this sort may influence the effectiveness of later feedings. Moreover, it has been suggested (Lipsitt,[16] p. 168) that such behavior when it occurs may be one of the earlier manifestations of infant operant conditioning.

I wish to conclude by calling attention to a primitive experiential effect, apparently of taste, upon the sucking behavior of the newborn child.[13] The data are actually from the same subjects as those from whom the frequency polygons for inter-suck intervals were drawn earlier in my presentation.[12] In that study, in addition to the groups which got either sucrose, distilled water, or no fluid throughout a 20-minute testing period, there were two other groups, both of which were alternated at 5-minute intervals between one condition and the other. In Figure 9–5 are shown the data of three groups of this experiment. One of them is the group that got sucrose administered for the 20-minute long period, one .02 ml drop of fluid for each suck, and another group which got water, one drop for one suck, throughout the same period. It can be seen that both the sucrose group and the water group maintained a stable level of sucking, in terms here of numbers of sucks per minute, with the sucrose group emitting slightly more sucks per minute than the water group.

Note now the behavior of the third group, which was alternated between sucrose and water, such that they

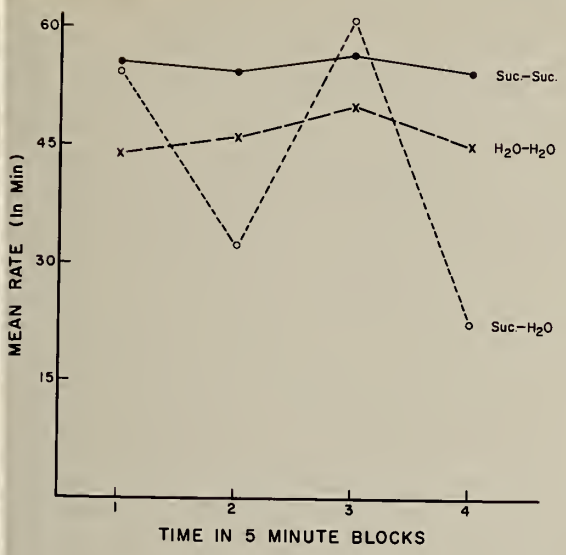


Figure 9–5. Mean sucking rate over four 5-min blocks for each of three groups, one receiving 15% sucrose throughout, one receiving water throughout, and the third alternating sucrose-water-sucrose-water in 5-min blocks. The newborn infants sucked less for contingently administered water when it followed a period of contingent sucrose presentation than when it followed a comparable period of water.

received sucrose within the first five minutes contingent upon sucking, water in the second five minutes, sucrose in the third five minutes, and water in the fourth five minutes. During sucrose-sucking, comparable numbers of sucks were invested by the alternated subjects relative to the sucrose-only group. During the two five-minute intervals in which they were required to suck for water, however, sucking rate fell off sharply, an apparent negative contrast effect indicating that the water was not as attractive to these infants following sucrose experience as it was to their peers who received only water throughout. This appears to be an early effect of experience, perhaps a rudimentary learning phenomenon. It is not unthinkable to suppose that infants who have a variety of sensory experiences within the first days and months of life might well be different in their later exploratory approaches to the sensory world and in their appreciation of novel experiences. It is rather an exciting supposition that the first days of the human being's life are not entirely a waste of time.

REFERENCES

1. Blanton, M.G.: The behavior of the human infant during the first thirty days of life. *Psychological Review,* 24: 456–483, 1917.
2. Bryan, E.S.: Variations in the responses of infants during the first ten days of post-natal life. *Child Development,* 1: 56–77, 1930.
3. Canestrini, S.: Ueber das Sinnesleben des Neugeborenen. *Monogr. Gesamtgeb. Neurol. Psychiat.,* Berlin: Springer, 1913, pp. 104.
4. DeLucia, C.A: A system for response measurement and reinforcement delivery for infant sucking-behavior research. *Journal of Experimental Child Psychology,* 5: 518–521, 1967.
5. Drummond, M.: *The dawn of mind: an introduction to child psychology.* London: Arnold, 1921.
6. Dubignon, J., and Campbell, D.: Sucking in the newborn in three conditions: non-nutritive, nutritive, and a feed. *Journal of Experimental Child Psychology,* 6: 335–350, 1968.
7. Eckstein, A.: Zur Physiologie der Geschmacksempfindung und des Saugreflexes bei Sauglingen. *Z. Kinderheilk,* 45:1–18,1927.
8. Gunther, M.: Instinct and the nursing couple. *Lancet,* 1: 575, 1955.
9. Gunther, M.: Infant behavior at the breast. In Foss, B., (Ed.), *Determinants of Infant Behavior,* London, Methuen and Co., Ltd., 1961, pp. 37–44.

10. Jensen,K.: Differential reactions to taste and temperature stimuli in newborn infants. *Genetic Psychology Monographs,* 12: 363–479, 1932.
11. Kaye, H.: Infant sucking behavior and its modification. In Lipsitt, L.P. and Spiker, C.C. (Eds.), *Advances in Child Development and Behavior,* II, New York, Academic, 1967, pp. 2–52.
12. Kobre, K.R.: A negative contrast effect in newborns, Brown University master's thesis, 1971.
13. Kobre, K.R., and Lipsitt, L.P.: A negative contrast effect in newborns. *Journal of Experimental Child Psychology,* 14: 81–91, 1972.
14. Kroner, T.: *Ueber die Sinnesempfindungen der Neugeborenen.* Breslau: Grass, Barth, 1881, p. 14.
15. Kussmaul, A.: *Untersuchungen uber das Seelenleben des Neugenborenen Menschen.* Tubingen: Moser, 1859, p. 32.
16. Lipsitt, L.P.: Learning in the first year of life. In Lipsitt, L.P. and Spiker, C.C. (Eds.), *Advances in Child Development and Behavior,* I, New York. Academic, 1963, pp. 147–195.
17. Lipsitt, L.P., Reilly, B.M., Butcher, M.J., and Greenwood, M.M.: The stability and inter-relationships of newborn sucking and heart rate. *Developmental Psychobiology.* 9: 305–310, 1976.
18. Lipsitt, L.P.: The synchrony of respiration, heart rate, and sucking behavior in the newborn. In Sinclair, J.C. and Warshaw, J.B. (Eds.), *Mead Johnson Symposium on Perinatal and Developmental Medicine,* No. 6, 1975, pp. 67–72.
19. Maller, O. and Desor, J.A.: Effect of taste on ingestion by human newborns. In Bosma, J.F. (Ed.), *Fourth Symposium on Oral Sensation and Perception,* Washington, Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 279–291.
20. Montagu, M.F.A.: *Prenatal Influences,* Springfield, Thomas, 1962.
21. Nowlis, G.: Taste elicited tongue movements in human newborn infants: an approach to palatability. In Bosma, J.F. (Ed.), *Fourth Symposium on Oral Sensation and Perception,* Washington, Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 292–303.
22. Peiper, A.: *Cerebral Function in Infancy and Childhood,* New York, Consultants Bureau, 1963.
23. Peterson, F. and Rainey, L.H.: The beginnings of mind in the newborn. *Bulletin of the Lying In Hospital City of New York,* 7: 99–122, 1910.
24. Pfaffmann, C.: The pleasures of sensation. *Psychological Review,* 67: 253–268, 1960.
25. Pratt, K.C.: The neonate. In Carmichael, L. (Ed.), *Manual of Child Psychology,* New York, Wiley, 1954, pp. 215–291.
26. Pratt, K.C., Nelson, A.K., and Sun, K.H.: The behavior of the newborn infant. *Ohio State University Studies, Contributions to Psychology,* Number 10, 1930.
27. Preyer, W.: *The Mind of the Child,* New York, Appleton (translated by H.W. Brown), 1882, translation in 1901.
28. Shinn, M.W.: Notes on the development of a child. *University of California Publications,* 1: p. 424, 1893–1899.
29. Shirley, M.M.: The first two years: a study of twenty-five babies. Volume II *Intellectual Development,* Minneapolis, University of Minnesota Press, 1933, p. 513.
30. Sonohara, T.: Systematic studies on psychology of human neonates: reactions to bitter stimuli. *Japanese Journal of Experimental Psychology,* 1: 127–141, 1934.
31. Steiner, J.: The human gustofacial response. In Bosma, J.F. (Ed.), *Fourth Symposium on Oral Sensation and Perception.* Washington, Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 254–278.
32. Stirnimann, F.: Le gout et 1' odorat du nouveau-ne. *Rev. franc. pediat.* 12: 453–485, 1936.
33. Weiffenbach, J.M. and Thach, B.T.: Elicited tongue movements: touch and taste in the newborn human. In Bosma, J.F. (Ed.), *Fourth Symposium on Oral Sensation and Perception.* Washington, Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 232–243.
34. Wolff, P.: The serial organization of sucking in the young infant. *Pediatrics,* 42: 943–956, 1968.

DISCUSSION

COLLIER: Does milk make the heart go faster?

LIPSITT: We don't know. We have some beginning evidence from a natural feeding situation where the mother breast-feeds the baby or a nurse feeds the baby milk from a bottle. Patrick Burke,[1] who is carrying out this work in my laboratory, sometimes finds that the heart rate goes down sharply when the fluid pours into the infant's mouth. A direct comparison with the automatic-nipple feeding situation which I just described, however, is unwarranted, for in the automatic situation the baby is getting a very tiny amount of fluid for each suck. The amount of fluid which the baby self-administers from a bottle nipple or from the breast is often very large, and the oral cavity fills up quickly. It seems that when babies get a large amount of fluid in their mouths contingent upon their sucking, heart rate often slows down drastically. I do not think that in those instances it is a result of taste. My supposition, entirely tentative at the moment, is that such feeding-induced bradycardia is actually a kind of fear response.

MALLER: Are there other things that make the heart beat faster besides the sweet taste?

LIPSITT: Well, of course, if you startle the baby—

MALLER: No, I am talking about taste.

LIPSITT: I see. I don't know that we want to say that distilled water has a taste, but when the baby simply sucks for distilled water, the heart rate goes higher than when he sucks for no fluid.

MALLER: What about other compounds like salts?

LIPSITT: I just don't know.

MALLER: What about acceptable compounds versus aversive ones?

LIPSITT: I just can't give you any information on that. We are just getting into the aversive stimulation kind of work now. I am quite certain that it will turn out to be a complicated matter, with heart rate acceleration occurring to some types of stimulation and heart rate deceleration to other types.

STEINER: What physiological explanation do you have for the elevation of heart rate?

LIPSITT: I leave that to the physiologists. Perhaps you can help with that. —I do not mean to slough off that question. It is a very important question. I have an explanation, but it is not a physiological explanation. I think that the infant slows down its sucking rate to savor the substance in its mouth. The increased savoriness of food in the mouth is exciting. I think it is a joy response, realizing that that is not very good physiology.

JOHNSON: There is the possibility of another explanation. I noticed that respiration was slower and more disturbed during sucrose ingestion.

LIPSITT: Yes. Perhaps that question can be held in mind for when Dr. Crook presents his findings. You will have an opportunity to see more of that sort of thing in his slides, for he will show you parametric data based upon several dilutions of sweet. When

[1] Burke, Patrick. Effects of formula- and breast-feeding on heart rate, sucking, and respiration in the newborn. Unpublished data. 1974.

the baby takes fluid into its mouth, in contrast to no-fluid sucking, there is increased respiratory disruption, but I don't think we could say, on the basis of information we have right now, that increasing the sweetness of the fluid disrupts respiration more.

LE MAGNEN: Have you tested the effects of hunger on these parameters?

LIPSITT: No, we have not done that yet. We are testing our babies midway between two feeds right now, so that they are "medium hungry." I can say, though, that we have done other work involving sucking and other eating-related responses (such as head-turning) in which hunger was varied, and this had no effect on at least the conditionability of the newborn. In such studies as we have run manipulating hunger, we have never exceeded the four-hour routine, so the infants are never terribly hungry when they appear in our experiments, even if they are maximally hungry as far as the hospital routine is concerned. We do know, on the basis of studies by former colleagues in my laboratory,[1,2] that deprivation does enhance non-nutritive sucking rate, and that there is a certain "work decrement" or satiation of sucking that occurs with protracted testing of sucking behavior within a session, but a great deal remains yet to be worked out as to the role of sweetness in all of this. It would be interesting to know whether increasing sweetness would enhance sucking during low-deprivation conditions or after sucking decrement due to fatigue.

[1] Levin, G.R. and Kaye, H. Nonnutritive sucking by human neonates. *Child Development*, 35:749–758, 1964.

[2] Levin, G.R. and Kaye, H. Work decrement and rest recovery during non-nutritive sucking in the human neonate. *Journal of Experimental Child Psychology*, 3: 146–154, 1966.

Chapter 10

THE DISCRIMINATION OF GLUCOSE AND WATER BY NEWBORN INFANTS

Trygg Engen

An earlier study [5] showed that the rate of sucking of two-day old infants varies with the substance: sucrose, glucose, or water. These results were in general agreement with similar studies [8,1] measuring volumes of intake in a feeding situation with regular nursing bottles. The present experiment was performed to extend the study of the effect of order of presentation of water and sugar. In our previous study, variation in order of presentation had yielded even larger differences in sucking rates than the variation in the ingested substance. It was suspected that the reinforcing property, presumably sweetness in this case, would depend on such positive and negative contrasts, (See ref. 7) as when changing from water to sugar and vice versa, and not solely on inherent reinforcing properties of the substance itself. Based on the earlier data, glucose at a concentration of 0.15 M was used since it seemed to be average in sweetness and also yielded typical results for sugar as compared with water.

The subjects were 16 boys and 16 girls who were full term single birth infants, ranging in age from one to three days. All of them had been judged to be normal and healthy by their pediatricians, and all of them were tested individually in the hospital with the consent of the mother.

The substances used were water and 0.15 M glucose. The infants' sucking was observed by means of a modified standard nipple designed to record each suck and deliver 0.02 ml fluid per suck, controlled by Digi-Bits programmer (See ref. 7). The measure was the number of sucks per minute recorded on a polygraph.

All infants were tested between 11:30 am and 1:00 pm, which is half-way between the infant's feeding periods. There were four experimental groups, two of them having been tested in the previous study.[5] In this case each infant was tested for 6 minutes with the glucose either before or after testing with water for 6 minutes. The groups added for the purposes of the present study were tested for the entire 12 minute period with either water or glucose. There were eight infants, four boys and four girls in each of these four groups, and each was assigned to a group at random according to a schedule.

Comparisons of the results for groups which switched from water to glucose and vice versa with the groups that were presented only one of these

Acknowledgment: The research was supported by a USPHS Research Grant to Lewis P. Lipsitt and by Grant Foundation funds to the Child Study Center, Brown University.

substances revealed no statistical evidence of either positive or negative contrast. Figure 10–1 illustrates the results. The group that received 0.15 M glucose for the entire period provided results nearly identical to the group that received this sugar concentration before water, and more crucially, also to the group that received the sugar after water. That is, there is no evidence of positive contrast. The group that received water throughout had a slightly higher rate of sucking, but not statistically different from the comparable groups that switched from water to glucose or vice versa. Only the difference between water and glucose was significant ($p<.005$, according to t-test). Finally, there is only an indication of the negative contrast, that has been obtained in other studies (See ref. 7). In the present experiment, infants receiving water after sugar sucked less, but not significantly less, for water than those who were presented it throughout the 12-minute period.

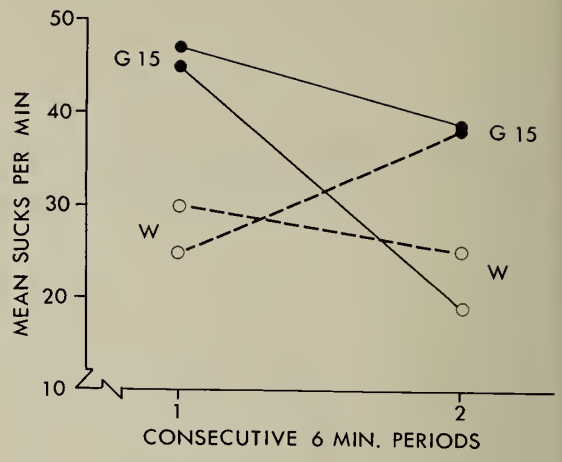


Figure 10–1. Mean number of sucks per minute as a function of duration of sucking for water and glucose at 0.15 M.

These results reveal a decrement over time but no differential effect associated with changing sapid substances mid-way through a feeding period. The preference for sweet over water appears to be quite reliable. By comparison, it seems that taste elicits more definite hedonic reactions than odors.[2,4] However, caution must be used in generalizing about an infant's hedonic experiences. One should not, as seems common, assume that he or she has preferences similar to that of an adult. For example, one cannot predict the odor preferences of young children using normative data obtained with adult observers.[3,6]

Furthermore, one should not take for granted that in the case of taste the infant has a scale of preferences ranging from tastes that are disliked through the point of neutrality or indifference to tastes, such as the sweet taste of sugar, that are liked. At the present time, there is only evidence that infants will suck more the sweeter the substance. It has not been demonstrated experimentally that he or she would in fact stop sucking and reject something unpleasant such as a bitter taste (See ref. 5). The presently available evidence does not make it possible to reject the hypothesis that, for the infant, there is only one side to the hedonic scale. Judged by what the infant will ingest, all tastes may be liked. Or is it the case that no taste will inhibit sucking?

REFERENCES

1. Desor, J.A., Maller, O. and Turner, R. Taste in acceptance of sugars by human infants. *Journal of Comparative and Psysiological Psychology,* 84: 496–501, 1973.
2. Engen, T.: The potential usefulness of sensations of ordor and taste in keeping children away from harmful substances. *Annals of the N.Y. Acad. Sciences,* 237: 224–228, 1974.
3. Engen, T. and Corbit, T.E.: Feasibility of olfactory coding of noxious substances to assure aversive responses in young children. Injury Control Research Laboratory Report ICRL-RR-60-6. U.S. Department of Health, Education and Welfare, 1970.
4. Engen, T. and Gasparian, R.E.: A Study of taste preferences in young children. Injury Control Research Laboratory Report ICRL-RR-73-2, 1973.
5. Engen, T., Lipsitt, L.P. and Peck, M. B.: Ability of newborn infants to discriminate sapid substances. *Developmental Psychology,* 10:741–744, 1974.
6. Engen, T. and Moskowitz, L.: The influence of trigeminal stimulation on children's judgments of ordor. Injury Control Research Laboratory Report ICRL-RR-71-4. U.S. Department of Health, Education and Welfare Publication No. (HSM) 72–10022, 1972.
7. Kobre, K.R. and Lipsitt, L.P.: A negative contrast effect in newborns. *Journal of Experimental Child Psychology,* 14:81–91, 1972.
8. Nisbett, R. and Gurwitz, S.: Weight, sex and the eating behavior of human newborns. *Journal of Comparative and Psysiological Psychology,* 73: 245–253, 1970.

Chapter 11

TASTE AND THE TEMPORAL ORGANIZATION OF NEONATAL SUCKING

C. K. Crook

The sucking response of the neonate has been a source of considerable interest to child psychologists. It has served as a dependent variable in experiments concerned with: 1) learning or the adaption of behavior to environmental contingencies,[6,22,23] 2) sensory skills,[15,17,28] and 3) social interaction.[8] In addition to its value for psychological assessment of the neonate, there is evidence that characteristics of the sucking rhythm also may serve to identify infants at risk and thus be of some diagnostic significance.[11,30]

Sucking behavior displays a remarkable degree of sensory motor coordination. Positive and negative pressure changes are generated by the activity of mouth, tongue and cheek and are integrated with respiratory and swallowing movements into a smooth rhythm. It is this very complexity of organization, coupled with the potentiality for objective recording, that has suggested sucking as a sensitive dependent variable and encouraged its application to studies of physiological and psychological functioning. However, examination of this literature indicates that quantification of the more subtle aspects of this behavior remains at a fairly primitive level—although this, in part, must be because the associated technical problems are great. For instance, relatively little attention has been directed toward isolating the variables influencing the amplitude of pressure changes generated during sucking. Neither is very much known of the variables that affect the frequency of swallowing. It has been generally the custom to record sucking as a single, all-or-none response. Thus, a record may be kept of the number of amplitude peaks or, if the measuring device is also equipped to deliver fluid at some critical response amplitude, [9,26] it may be simply the number of such "criterion" sucks that is recorded.

It is likely that for many purposes, this treatment of the response will prove quite adequate. Many studies that have simplified the sucking rhythm in this way have then gone on to take total number of responses per unit time as a dependent variable. Where this has been the practice, the data effectively has been impoverished yet further, for such measures of response rate ignore the detailed temporal organization of the, now digitized, sucking rhythm. Again we may suppose that many aims are well

served by measurements no more sophisticated than the enumeration of responses within a given period of time. However, it is potentially of interest to consider how changes in response rate are mediated by changes in the underlying temporal organization of responding. Indeed, at least one study [27] has demonstrated how such detailed measures of the sucking pattern proved sensitive to the effect of an experimental manipulation where a simple record of overall response rate did not.

Wolff [30] referred to the temporal organization of sucking as its "microstructure" and he has also expressed concern that relatively little attention has been directed towards its elaboration. Although a number of papers have appeared recently that serve to rectify this omission,[5,7] it remains true that we have only limited knowledge of how the serial organization of sucking is modified.

An earlier study by Balint [1,2] is perhaps the first to give detailed consideration to this microstructure. Balint recognized, as indeed had others before him, that sucking is often organized as a sequence of response bursts separated by resting periods or pauses. His data suggest a distinction I shall pursue in this paper. With respect to its temporal organization, sucking can be analyzed first in terms of a *coarse structure* and then further in terms of a *fine structure.*

The consideration of coarse structure focuses on the response burst as the unit of analysis. Within a defined period, we can describe how many of such bursts occur, what is their average length and what is the average pause duration between them. Evidently, the concept of a response burst necessitates a decision as to the minimum inter-suck interval that will define a pause. This problem has been discussed by Kaye.[16]

In considering fine structure, the temporal separation of sucks within bursts is taken as a unit of analysis. It might be of interest, for instance, to examine how an experimental treatment affected the average value of such inter-response times (IRTs) or even the serial distribution of IRTs within bursts. The average IRT is, of course, essentially a transformation of the within burst, or "local" response rate. Balint [2] referred to this as the "basic frequency," N_1.

Both the coarse and fine structure of sucking is known to be affected by whether or not fluid is made contingent upon individual sucks. Nonnutritive sucking has a more marked burst and pause rhythm than nutritive sucking and IRTs are typically shorter.[29,30] More detailed information concerning how the quality and quantity of response-contingent fluid affects the organization of nutritive sucking is lacking. The study reported here examines how sucrose solutions differing in concentration modify the sucking pattern.

Several experiments have been reported in which solutions differing in sweetness have been compared with respect to their effects upon sucking. Dubignon and Campbell [12,13] have compared sucking for a 5% dextrose solution with milk formula bottle feeding and breast feeding. However, these experiments are complicated by the fact that the dextrose was delivered only after every tenth suck. Evidently, no conclusions about the effects of sweetness *per se* could be drawn from these studies. This is also

true for a similar experiment performed by Kron, Stein, Goddard and Phoenix.[20]

A more systematic approach to the question of sweet taste sensitivity in newborn infants can be found in the work of Engen, Lipsitt and Peck,[14] Desor, Maller and Turner [10] and Nisbett and Gurwitz.[25] In these experiments, sugar solutions of various concentrations were presented to groups of newborns and the total quantity of fluid ingested, or the total number of sucks made, was recorded. The results obtained in each case demonstrate discrimination on the dimension of sweetness. The present experiment extends these findings by examining how changes in the temporal organization of sucking mediate the differences in intake. In addition, this experiment uses a range of sucrose concentrations that includes a value greater than any used previously and also examines the effects of the sweetness variable across the course of a longer feeding period.

METHOD

Subjects

The subjects were 35 female and 40 male infants between 48 and 72 hours of age. Seven additional infants were tested but rejected from the study for failing to satisfy the "baseline" sucking criterion described below. Subjects were arbitrarily allocated to one of 5 experimental groups and tested between 11:30 a.m. and 1 p.m. The previous feed by the mother had occurred at 9 a.m. All subjects were bottle fed during their normal feedings.

Apparatus

The feeding apparatus has been described in detail elsewhere by DeLucia [9] and Kobre and Lipsitt.[19] Sterile polyethylene tubes were pulled through two small holes punched in a standard blank Davol nipple. One of these tubes ran to a transducer that converted negative pressure changes into electrical signals that were sent to one amplifier of a polygraph recorder. The amplifier permitted the experimenter to specify an amplitude criterion that defined a suck. Signals that reached this criterion were sent in the form of pulses to solid state programming equipment. In this way, a criterion suck activated a timer which then pulsed the fluid pump driver causing 0.020 ml. of fluid to be delivered through the second tube inserted in the nipple.

Each criterion suck also caused a brief tone pulse to be recorded onto one channel of a stereo tape recorder. The tapes were later played back through the analog channels of a PDP 12 computer. The computer recorded the temporal separation of these pulses in 0.05 second units and stored them on magnetic tape for subsequent analysis. Subjects were tested in a crib housed in a 6′ x 7′ x 11′ sound attenuated chamber. Background white noise was played into this chamber at an intensity of 74 db as determined at the position of the infant's head.

Procedure

Individual infants were brought to the laboratory by a nurse who attached electrodes for monitoring

heart rate. Each subject was swaddled and placed on his left side. The nipple, supported by a cushion, was placed in the subject's mouth.

If, upon insertion of the nipple, the subject failed to suck at once, encouragement was given by gentle stroking of the cheek. Sucking during the first 2 minutes did not result in the delivery of fluid. The purpose of this period was to enable the experimenter to calibrate the equipment and establish a sucking amplitude criterion. Adjustments were made to the sensitivity of the amplifier until sucks were causing a full 5 cm. deflection of the pen. The amplitude criterion for recording the occurrence of a suck was fixed at a 2.5 cm. pen deflection. Subjects emitting less than 30 sucks in this period (seven subjects) were eliminated from the study.

Sixty subjects were arbitrarily allocated to four groups of equal size differing with the respect to the fluid delivered following a criterion suck. Group 15 received a 15% (0.44M) sucrose solution, Group 10 a 10% solution (0.29M), Group 5 a 5% solution (0.15M), and Group 0 a 0% solution (distilled water). An additional 15 subjects were allocated to a fifth group (NN) and received one minute of 5% sucrose after which the fluid delivery mechanism was turned off. The experimental period for this group then began and subjects were allowed to suck non-nutritively.

The maximum duration of the experimental period was 20 minutes. A session could be terminated early if: 1) a period of 3 minutes elapsed during which no sucking occurred, or 2) the nipple was ejected from the mouth more than once. Following the first such ejection the nipple was gently replaced.

RESULTS

Table 11–I shows how session durations were influenced by the various experimental conditions and gives, for each group, the number of subjects that were tested for the maximum possible period of 20 minutes.

The results presented below describing the performance of the four nutritive groups have been taken from the first 12 minutes of testing. All subjects remained in the experiment for at least this length of time. Results from the non-nutritive group

TABLE 11–I

MEAN AND MEDIAN SESSION LENGTHS FOR SUBJECTS IN EACH OF THE FIVE EXPERIMENTAL GROUPS AND NUMBER OF SUBJECTS TESTED FOR THE MAXIMUM OF 20 MINS.

	Group				
	NN	0	5	10	15
Mean session length	12.3	15.4	19.2	18.0	18.5
Median session length	11.7	17.3	19.4	19.0	19.6
# of Ss tested for 20′	6	7	14	11	14

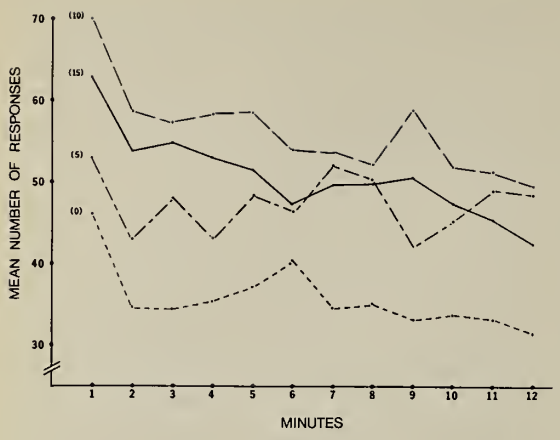


Figure 11–1. Mean number of responses per minute plotted across the first 12 minutes of the feed for the four nutritive groups.

are based upon only the first 4 minutes of testing but similarly allow the inclusion of all Group NN subjects for analysis.

Total amount of sucking

Figure 11–1 shows for each nutritive group the mean number of sucks emitted in each consecutive minute of the feed. An analysis of variance on this data gives an effect of concentration that is significant beyond the 1% level ($F=6.9$, $df=3/56$) and a minutes effect also significant beyond this level ($F=5.8$, $df=11/616$). The inversion of Groups 10 and 15 should be noted.

Time units of one minute are short, particularly, for a consideration of the detailed temporal organization of the sucking rhythm as individual bursts and pauses may exceed one minute in length. Consequently, within session effects were assessed by dividing the 12 minutes into three 4 minute "periods."

In the top diagram of Figure 11–2 (R), the mean number of responses emitted in a four minute period by subjects in each of the four nutritive groups is shown. Periods is the parameter on this graph. Thus, a heavy line describes the function during the first 4 minutes, a dashed line during the second 4 minutes and a dotted line during the third 4 minutes. This convention is followed in the lower diagram showing the effect of sucrose concentration on the cumulative pausing time (PT) during successive periods of the feed. For purposes of this study, a pause was defined as any inter-suck interval greater than 3 seconds. Examination of the frequency distributions of all inter-suck intervals greater than 1

TABLE 11–II

F—RATIOS FOR CONCENTRATION PERIOD AND INTERACTION EFFECTS FROM ANALYSIS OF VARIANCE FOR SUCKING PARAMETERS

Parameter	*Concentration*	*Periods*	*Concentration x Periods*
	(df = 3/56)	*(df = 2/112)*	*(df = 6/112)*
Mean number of responses (R)	8.8***	7.3***	2.0
Cumulative pausing time (PT)	11.5***	9.6***	2.9*
Inter-response time (IRT)	4.3**	3.8**	1.5
Inter-burst interval (IBI)	5.4**	0.1	0.9
Number of bursts (NB)	7.9***	1.6	4.9***
Number of responses per burst (BL)	7.9***	6.6**	2.6*

Notes: See Figures 11–2 and 3 for graphic presentation of these data
* = $p<.05$
** = $p<.01$
*** = $p<.001$

second suggested that this was a suitable choice.*

The top rows of Table 11–II describe the results of an analysis of variance performed on the data of Figure 11–2. The effects of both concentration and periods are highly significant. A significant interaction term for the PT data prompted separate tests on main effects and these results are given in the top row of Table 11–III. The concentration effect is significant in all three periods and Groups 10 and 15 show a significant increase in PT across periods. Although it might be expected that the R and PT functions would be mirror images, such an outcome will depend on whether the experimental treatments differentially modify what I have termed the fine structure of sucking—and it will become apparent that they do.

Coarse structure

Figure 11–3 is constructed in a similar manner to Figure 11–2 and illustrates the effects of sucrose concentration on four parameters of the sucking rhythm during successive periods

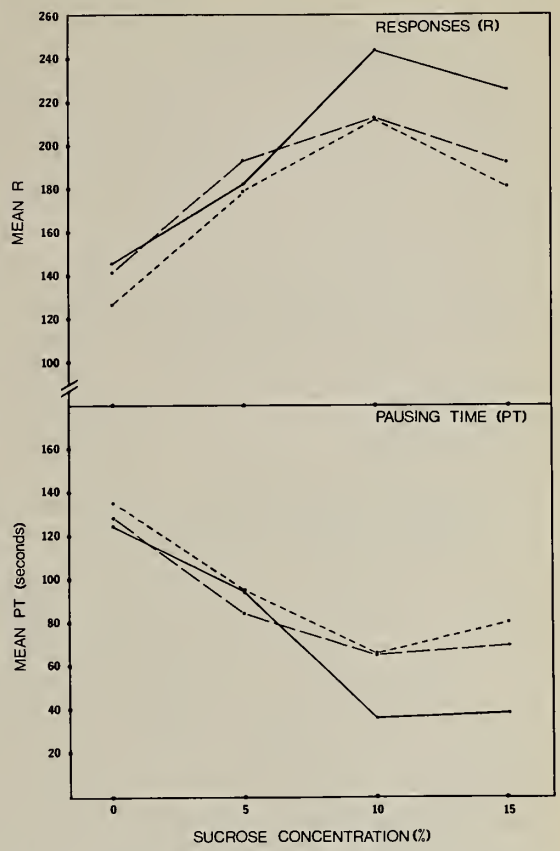


Figure 11–2. The upper diagram shows the mean number of responses made as a function of sucrose concentration. The lower diagram shows the mean cumulative pausing time as a function of sucrose concentration. "Period" is the parameter in both diagrams. The solid line referring to the first 4 minutes of the feed, the dashed line the second 4 minutes, and the dotted line the third 4 minutes.

*The experimental data were reanalysed taking a 5 second pause criterion. The pattern of results was essentially identical to that reported here.

TABLE 11–III

F—RATIOS FOR THE SEPARATE CONCENTRATION AND PERIOD MAIN EFFECTS FOR THE SUCKING PARAMETERS THAT YIELD SIGNIFICANT CONCENTRATION X PERIOD INTERACTIONS.

Parameter	*Period (df = 3/56)*				*Concentration (df = 2/112)*		
	0%	5%	10%	15%	1	2	3
Cumulative pausing time (PT)	0.6	0.8	6.3***	10.7***	14.9***	6.4***	7.1***
Number of bursts (NB)	5.5**	1.4	3.4*	5.6**	7.8***	1.0	2.4
Number of responses per burst (BL)	0.1	0.4	9.6***	4.1*	12.5***	2.5	2.2

Notes See Table 11—II
* = $p<.05$
** = $p<.01$
*** = $p<.001$

of the feed. The three parameters reflecting coarse structure are: 1) number of bursts (NB), 2) burst length or the mean number of responses per burst (BL), and 3) pause length or inter-burst interval (IBI). The top two diagrams in Figure 11–3 and the bottom right diagram show group means for these parameters.

The results of analyses of variance on these data are given in Tables 11–II and III. Sucrose concentration has a significant effect upon IBI, shorter pauses being associated with the higher concentrations. However, for a given nutritive condition, IBIs do not change across the three periods considered. For both the NB and BL parameters there is a significant concentration x periods interaction. Reference to Table 11–III indicates that in both cases the concentration variable exerts an effect only during the first period. During this time there appear to be fewer and longer bursts associated with the higher concentration solutions. Both Groups 10 and 15 demonstrate a significant periods effect for NB and BL. The general tendency is for there to be more but shorter bursts as the session proceeds. Subjects in Group 0 tend to emit fewer bursts later in the session but their length remains stable.

Fine structure

As specified above, "fine structure" refers to the rates of responding within bursts. The term interresponse time (IRT) is used to describe the time between two sucks within a burst. Thus, given the present definition of a pause or IBI, no IRT can be greater than 3 seconds.

The bottom left diagram of Figure 11–3 shows the effects of sucrose concentration upon IRTs during the three periods of the experiment. Reference to Table 11–II indicates that both the concentration and periods effects are significant. The IRTs increase in length as sucrose concentration is increased. This effect is less evident later in the feed.

The analysis of IRTs may be extended further through a consideration of how their duration changes at different points within a burst. For each subject, the means of the first eight IRTs from all bursts emitted in the first 12 minutes of testing and containing 12 or more responses were calculated. From this sample of bursts, the means of the last 8 IRTs were calculated. Group results are presented in Figure 11–4. The right hand set of curves in this figure show mean IRT as a function of ordinal position in the bursts, i.e., the point above 1 on the ordinate specifies the mean of the first IRT and that above 2 the mean of the second IRT and so on. The curves on the left show mean IRTs at the end of bursts. Here, the point above 1 specifies the mean of all the last IRTs in the sampled bursts, the point above 2 then being the mean of all the penultimate IRTs and so on back towards the start of the burst. The results from the four nutritive groups are presented, along with those from the non-nutritive group for comparison. Analyses of variance was performed on data from the nutritive groups. Considering first the burst onset data, the effect of concentration is not significant ($F=1.6$, $df=7/56$), the effect of ordinal position of the IRT is significant ($F=16.5$, $df=7/393$, $p<0.01$) and there is a sig-

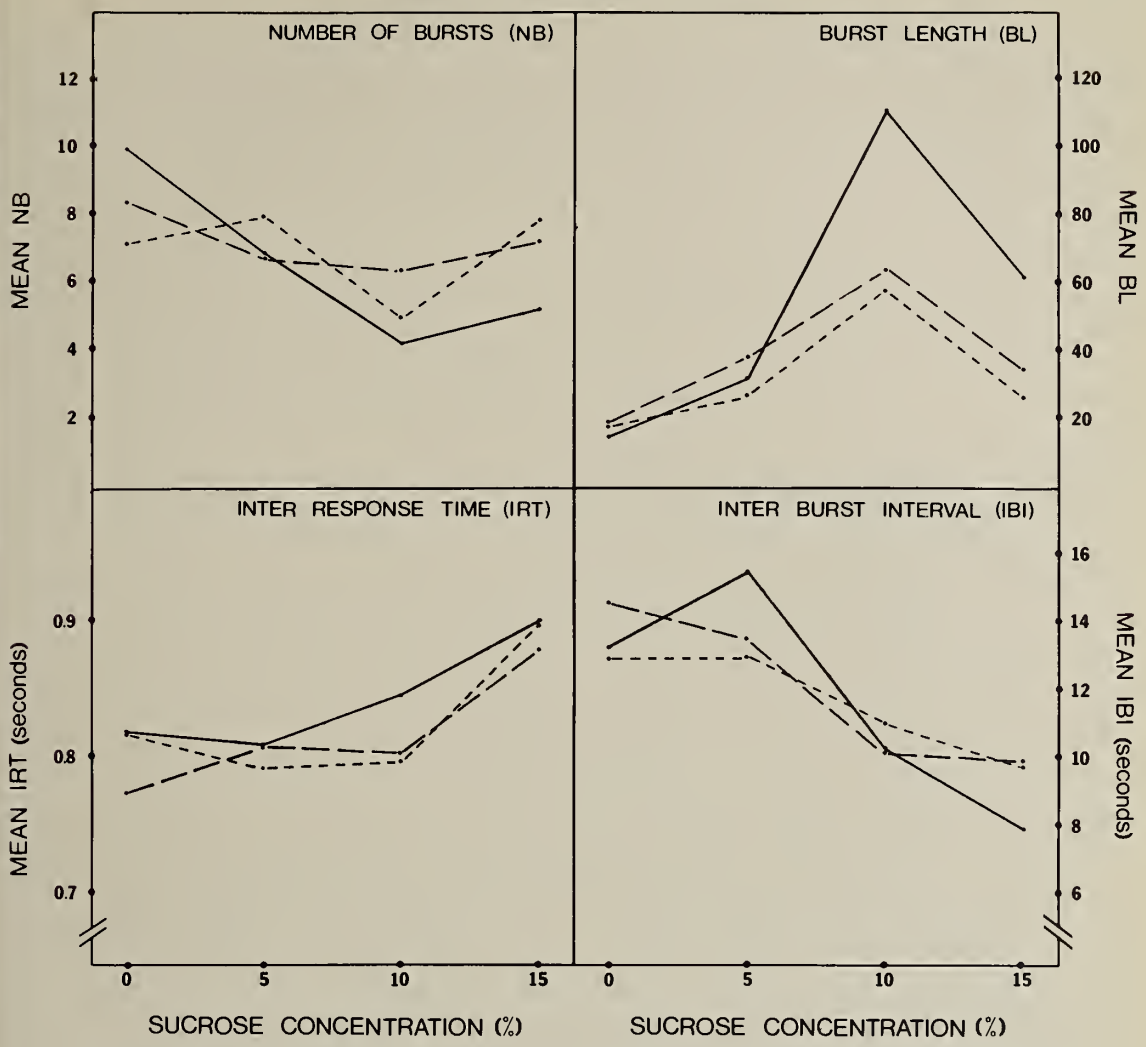


Figure 11–3. Changes in the mean value of various measures of the sucking rhythm as a function of sucrose concentration. See text for a definition of these measures. "Period" is again the parameter in all diagrams.

nificant interaction of these terms ($F=1.7$, $df=21/393$, $p<0.05$). Separate tests on the main effects of the concentration at different ordinal positions of the IRT show that only the differences at positions 2 and 3 are significant (respectively, $F=3.0$, $p<0.01$; $F=3.4$, $P<0.01$; $df=3/56$). Tests of the main effects of ordinal position of an IRT given different concentrations show significant F ratios for Group 0 ($F=10.4$, $df=7/393$, $p<0.01$), Group 5 ($F=6.5$, $df=7/393$, $p<0.01$) and Group 15 ($F=3.7$, $df=7/393$, $p<0.01$). The ordinal position effect is not significant for Group 10.

For IRTs from the end of bursts, concentration does not exert a significant effect ($F=1.3$, $df=3/56$) while the effect of ordinal position of the IRT is significant ($F=13.1$, $df=7/393$, $p<0.01$).

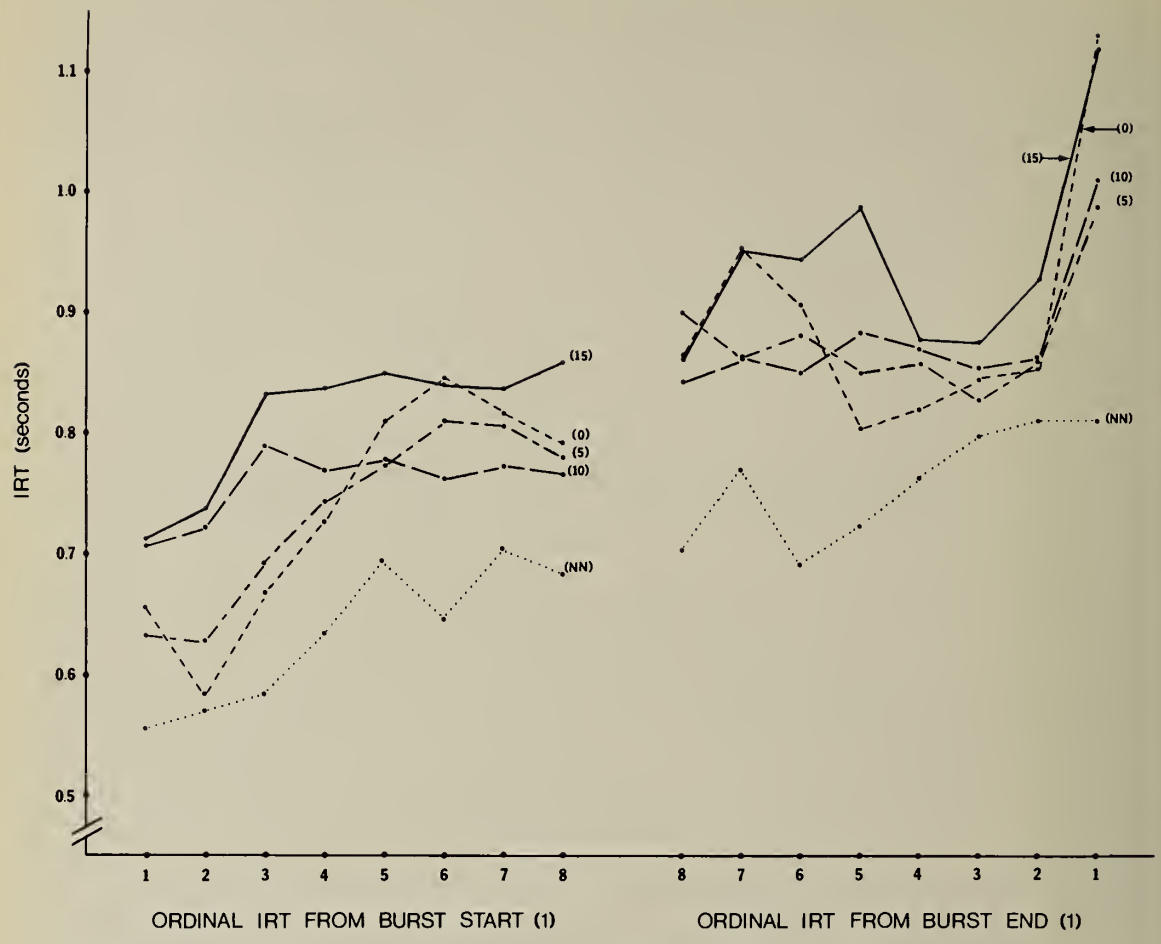


Figure 11–4. The left hand diagram shows mean IRT as a function of ordinal position from the start of burst. The diagram on the right shows mean IRT as a function of ordinal position from the end of a burst. Data is taken from bursts comprising 12 or more responses. The results from nutritive and non-nutritive conditions are shown.

DISCUSSION

The data presented in Figures 11–1 and 2 indicate a significant effect of sucrose concentration on both the number of sucks emitted and the cumulative pausing time. The effect is most dramatic during the early part of the feed; a finding that suggests further studies of neonatal taste sensitivity that are based upon intake measures would do best to use short feeding periods, perhaps in the order of 3 or 4 minutes.

The preference ranking suggested by visual inspection of Figures 11–1 through 3 is $0<5<15<10$. A series of Newman Keuls tests were performed on the means of the various sucking parameters during the first period and the $15<10$ ordering is supported statistically at the 5% level only by the

BL measure. Nevertheless, it is of interest to note that the functions presented by Desor *et al* [10] also suggest a preference asymptote is approached at their most concentrated sugar (~10% sucrose).

Analysis of the coarse structure of sucking rhythm indicates that the higher response rate maintained by sweeter sucrose solutions is mediated by fewer but longer bursts separated by shorter pauses. The effect of the concentration variable is lessened as the feed progresses, largely as a consequence of a decrease in burst lengths. The length of IBIs would appear to be determined exclusively by the concentration variable; this parameter contributes little to the decline in response rate across the feed.

With regard to the fine structure of the response, Wolff [29] has claimed that following a change in the composition of the fluid delivered "the mean peak interval between sucks of an uninterrupted sequence does not change" (p. 357). A change is, however, apparent in the present data. Sweeter fluids tend to maintain slower within burst responding.

Such an effect has been reported on a within subject basis elsewhere.[18] Lipsitt [24] has noted the paradoxical nature of this finding. Why should an organism respond more slowly for something, which on the basis of other observations we suppose that it prefers? He has suggested that this paradox might be resolved by viewing the slower sucking rates for sweeter solutions as reflecting a process of "savouring." This is an attractive hypothesis. However, examination of the present data will indicate that by the third period of the session most sucking parameters fail to differentiate Groups 5, 10 and 15. Yet the mean IRT of subjects in Group 15 remains consistently longer than that of the other two groups throughout the session. If slower within burst responding reflects "savouring" and thus is to be regarded as an index of preference, it is necessary to recognize that this measure does not always covary with those derived from records of intake or overall response output.

The effect of sugar concentration upon IRTs does merit further study. It is possible for instance, that the slower responding is in fact more vigorous responding; sweeter fluids may lead to higher amplitude sucking. It should be borne in mind that more concentrated sucrose solutions have higher viscosities. This is a variable that so far has been left uncontrolled in studies of infant taste and it is one that may be influential in the IRT results discussed here.

IRTs were also analysed with respect to their ordinal positions within bursts. IRTs were typically short at the start of a burst, lengthening to a stable duration across the first four or five ordinal response positions. Wolff [30,31] has observed decelerations of sucking rate at the onset of bursts that are similar to those shown in Figure 11–4. Although he failed to find such an effect during nutritive sucking [30] (See p. 946), it is clearly obtainable under the present conditions. There is also some suggestion that the extent of these decelerations is related to sucrose concentrations. The more preferred fluids show the effect less dramatically. It would thus appear that sweetness can affect the fine structure of sucking not only at the level of the mean IRT but also at the

level of patterning within bursts.

That sweetness should affect the fine structure of sucking at all is of some importance with regard to the choice of a dependent variable in taste preference studies with neonates. The measure R presented in Figure 11–2 fails to differentiate Groups 5 and 15 during the first period (a *post hoc* Newman Keuls test gives $p>0.05$), while PT does differentiate these groups ($p<0.05$). Although the two groups may emit a comparable number of responses in a four minute period, subjects in Group 15 require a longer portion of that period to do so as they suck more slowly. Time spent pausing is, at this point, more sensitive to the group differences.

We have demonstrated that sweetness modifies both the coarse and fine structure of sucking. These effects may interact in such a way as to reduce the sensitivity of simple intake measures of preference or measures based upon a count of sucks. A further understanding of why sweeter sucrose solutions support not only more sucks per unit time but also slower sucking is evidently required. At present the importance of detailed examination of the effects of stimuli on the organization of sucking must be stressed.

A point should be raised concerning the 0% sucrose or distilled water group. The amount of sucking by subjects in this group remained consistently less than that of subjects in the other nutritive groups which, by the end of the feed, were themselves essentially comparable. The experiments of Bartoshuk and her colleagues [3,4] have demonstrated that after the adult tongue is adapted to weak sodium chloride solutions, subadapting concentrations taste bitter. It is known that infants have particularly high sodium levels in their saliva.[21] Samples taken in our laboratory indicate concentrations of sodium in the mixed saliva of neonates can be as high as 80 mEq $Na^+/1.$, while adult levels are usually in the region of 10 mEq $Na^+/1$. This suggests that distilled water may taste bitter to the neonate. We should at least be cautious in assuming that subjects in Group 0 were sucking for a neutral or tasteless fluid.

Finally, the relevance of the present results to the distinction between nutritive and non-nutritive sucking should be noted. Wolff [30] has summarized the structural difference thus: ". . . nutritive sucking is organized as a continuous stream rather than an alternation of bursts and rest periods; and, its mean rate per second is slower and usually about half that of non-nutritive mouthing" (p. 948). This amounts to differences in both what I have termed the coarse and fine structure of the response. Unpublished data from our laboratory indicate that nutritive sucking can appear more similar to non-nutritive sucking as the amount of fluid contingent upon each suck is reduced. That is, with small amounts of fluid, sucking has a more marked burst and pause pattern and IRTs are shorter. The data reported here suggest that not only quantity but also quality of the nutrient—in this case its sweetness—can alter the nutritive sucking pattern to resemble that characteristic of non-nutritive conditions. Thus, sucking for 5% sucrose is characterized by shorter IRTs and more pauses than sucking for 15% sucrose—at least in the early portion

of a feed. The dichotomy of sucking modes is perhaps an oversimplification; continuity and speed of response can be apparent to varying degrees during nutritive sucking. One variable influencing both is the sweetness of the nutrient.

REFERENCES

1. Balint, M.: Individual differences of behavior in early infancy, and an objective method for recording them: I. Approach and the method of recording. *Journal of Genetic Psychology,* 73: 57–79, 1948.
2. Balint, M.: Individual differences of behavior in early infancy, and an objective method for recording them: II Results and conclusions. *Journal of Genetic Psychology,* 73: 81–117, 1948.
3. Bartoshuk, L.M.: Water taste in man. *Perception and Psychophysics,* 3: 69–72, 1968.
4. Bartoshuk, L.M., McBurney, D.H., and Pfaffmann, C.: Taste of sodium chloride solutions after adaption to sodium chloride: Implications for the "water taste." *Science,* 178:988–990, 1972.
5. Bosack, T.N.: Effects of fluid delivery on the sucking response of the human newborn. *Journal of Experimental Child Psychology,* 15: 77–85, 1973.
6. Brown, J.: Instrumental control of the sucking response in human newborns. *Journal of Experimental Child Psychology,* 14: 66–80, 1972.
7. Brown, J.: Non-nutritive sucking in great ape and human newborns: some phylogenetic and ontogenetic characteristics. In Bosma, J. F. (Ed.): *Fourth Symposium on Oral Sensation and Perception: Development in the Fetus and Infant.* Washington, D. C., Superintendent of Documents, U. S. Government Printing Office, 1973, pp. 118–131.
8. Campbell, D.: Sucking as an index of mother-child interaction. In Bosma, J.F. (Ed.): *Fourth Symposium on Oral Sensation and Perception: Development in the Fetus and Infant.* Washington, D.C., Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 152–165.
9. De Lucia, C.A.: A system for response measurement and reinforcement delivery for infant sucking behavior research. *Journal of Experimental Child Psychology,* 6: 518–521, 1967.
10. Desor, J., Maller, O., and Turner, R.: Taste in acceptance of sugars by human infants. *Journal of Comparative and Physiological Psychology,* 84: 496–501, 1973.
11. Drier, T., and Wolff, P.H.: Sucking, state, and perinatal distress in newborns. *Biology of the Neonate,* 21: 16–24, 1972.
12. Dubignon, J., and Campbell, D.: Sucking in the newborn in three conditions: Non-nutritive, nutritive and a feed. *Journal of Experimental Child Psychology,* 6: 335–350, 1968.
13. Dubignon, J., and Campbell, D.: Sucking in the newborn during a feed. *Journal of Experimental Child Psychology,* 7: 282–298, 1969.
14. Engen, T., Lipsitt, L.P., and Peck, M.B.: Ability of newborn infants to discriminate sapid substances. *Developmental Psychology,* 10: 741–744, 1974.
15. Haith, M.M.: The response of the human newborn to visual movement. *Journal of Experimental Child Psychology,* 3: 235–243, 1966.
16. Kaye, H.: Infant sucking behavior and its modification. In Lipsitt, L.P., and Spiker, C.C. (Eds.): *Advances in Child Development and Behavior,* New York, Academic Press, 1967, pp. 1–52.
17. Keen, R.: Effects of auditory stimuli on sucking behavior in the human neonate. *Journal of Experimental Child Psychology,* 1: 348–354, 1964.
18. Kobre, K.R.: A negative contrast effect in newborns. Unpublished master's thesis, Brown University, 1971.
19. Kobre, K.R., and Lipsitt, L.P.: A negative contrast effect in newborns. *Journal of Experimental Child Psychology,* 14: 81–91, 1972.

20. Kron, R.E., Stein, M., Goddard, K.E., and Phoenix, M.D.: Effect of nutrient upon the sucking behavior of newborn infants. *Psychosomatic Medicine,* 29: 24–32, 1967.
21. Lawson, D., Saggers, B.A., and Chapman, M.J.: Screening for cystic fibrosis by measurement of unstimulated saliva solium levels. *Archives of Disease in Childhood,* 42: 689–690, 1967.
22. Lipsitt, L.P., and Kaye, H.: Conditioned sucking in the human newborn. *Psychonomic Science,* 1: 29–30, 1964.
23. Lipsitt, L.P., Kaye, H., and Bosack, T.: Enhancement of neonatal sucking through reinforcement. *Journal of Experimental Child Psychology,* 4: 163–168, 1966.
24. Lipsitt, L.P.: Taste in human neonates: Its effects on sucking and heart rate. This symposium.
25. Nisbett, R., and Gurwitz, S.: Weight, sex and the eating behavior of human newborns. *Journal of Comparative and Physiological Psychology,* 73: 245–253, 1970.
26. Sameroff, A.J.: An apparatus for recording sucking and controlling feeding in the first days of life. *Psychonomic Science,* 2: 355–356, 1965.
27. Sameroff, A.J.: Nonnutritive sucking in newborns under visual and auditory stimulation. *Child Development,* 38: 443–452, 1967.
28. Semb, G., and Lipsitt, L.P.: The effects of acoustic stimulation on cessation and initiation of non-nutritive sucking in neonates. *Journal of Experimental Child Psychology,* 6: 585–597, 1968.
29. Wolff, P.H.: Sucking patterns in infant mammals. *Brain Behavior and Evolution,* 1: 354–367, 1968.
30. Wolff, P.H.: The serial organization of sucking in the young infant. *Pediatrics,* 42: 943–956, 1968.
31. Wolff, P.H.: The interaction of state and non-nutritive sucking. In Bosma, J.F. (Ed.): *Third Symposium on Oral Sensation and Perception,* Springfield, C.C. Thomas, 1972, pp. 293–312.

DISCUSSION

STEINER: Is my recollection of your apparatus correct, in that after each suck the baby receives a pre-set amount of fluid independent of how he sucks? Would not your recordings and those of Drs. Lipsitt and Engen then refer to the frequency of movements carried out by the baby and not to the consumption of fluid? In your apparatus the pre-set amount of fluid does not vary with other parameters of sucking. If that would be the case, it would perhaps reflect the more natural conditions of sucking during a feed.

CROOK: Yes. All of our results describe the temporal organization of sucking responses and what we count as a response is any negative pressure change causing a pen sweep of a pre-set amplitude on the polygraph record. During the baseline period on non-nutritive sucking the amplitude gain is set such that only the occasional suck made at the start of a burst fails to reach the criterion.

I am not trying to simulate the conditions that characterize a normal feed. Infants who have been fed these thin sugar solutions from a commercial bottle in our laboratory have often shown signs of distress including apnoea and bradycardia, and in the process quite a lot of fluid may be spilled. With our nipple the infant is comfortable and the small amounts of fluid delivered leave us less concerned with the effect of post-ingestional factors. Because that amount is fixed at each suck, my results, and those of

Drs. Lipsitt and Engen, can easily be transformed from "number of responses" to "millilitres consummed." However, I specifically wanted to show changes in the serial organization of the response induced by taste stimuli.

STEINER: I am concerned about your reliance on fluctuations in the number of sucks as a reliable indicator of hedonic response. Doesn't one have to be very careful in using a simple increase in the number of sucks as a direct indication of pleasantness?

CROOK: We have observed both suppression and potentiation of sucking by stimuli from modalities other than taste. Increases in sucking frequency that accompany such stimulation have been identified in the past both with orienting and defensive reactions. Certainly that area of research might be clarified if the effects of a given stimulus on the sucking response was related to its effects on, say, heart rate, respiration or facial expression. Characterizing some aspect of the neonate's behavior as an hedonically positive or negative response is a difficult and dangerous game—although it does seem to me to be something that is very important for psychologists to try and do. For my part, I admit the assumption in my experiment that more sucking means greater preference. This experiment does involve a contingency between the response and the stimulation and when an increase in responding is observed within the context of such a contingency I think my assumption is then not unreasonable. At least I think this reflects beliefs widely shared among psychologists. However, I have certainly been reassured by the findings that Dr. Steiner has reported using the facial response.

COLLIER: I can just add one comment. If you look at lick rate in rats drinking saccharin the pattern of licks is different from that for sugar. The local rate is higher and the bursts are shorter

CROOK: Oh, I was under the impression that licking, the consummatory behavior of the adult rat, was essentially constant for any substance.

COLLIER: Not with saccharin. We have observed higher rates with noxious substances. Otherwise, you seem to get a relatively constant average rate of seven per second. Initial rates are higher and terminal rates lower.

CROOK: You're talking about a rate within an episode.

COLLIER: No, the rate within a single burst of responses.

NOWLIS: I should add a few comments on the question of the newborn's response to aversive tastes which Dr. Engen raised earlier. I have given a number of infants 10^{-4} Molar quinine hydrochloride while monitoring pressures from the front and back of the tongue with a specially constructed pressure transducing nipple. As I reported earlier,[1] this concentration of quinine invariably interrupts a sucking bout after the first few sucks. If the nipple is left in the mouth thereafter, the infant generally produces movements of the posterior tongue which tend to push the nipple out of the mouth. A similar pattern was present in some infants offered isotonic saline; in other infants, isotonic or hypotonic saline yielded a

[1] Nowlis, G.H.: Taste-elicited tongue movements in human newborn infants: An approach to palatability. In Bosma, J.F. (Ed.) *Fourth Symposium on Oral Sensation and Perception.* Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 292–303.

mild ingestion response. This variability of response to salt may be due to the great variability among newborn infants of salivary sodium concentration.[1]

Crook: I have actually found a negative correlation between water intake and salivary sodium in neonates and children in the first two months of life. Alas, the correlation fell short of statistical significance. However, I am sure you appreciate that there are a lot of problems attached to the accurate measurement of salivary sodium —particularly in young children. My results do suggest the relation might warrant more rigorous study.

[1] Prader, A., Gautier, E., Gautier, R., Naf, D., Semer, J. M., & Rothschild, E. J. The Na and K concentration in mixed saliva: influence of secretion rate, stimulation, method of collection, age, sex, time of day, and adrenocortical activity. In Wholstenholme, G.E.W., and Cameron, M.P. (Eds.) *The Human Adrenal Cortex,* London, Churchill, 1955, p. 382.

Chapter 12

PREFERENCE FOR SWEET IN HUMANS: INFANTS, CHILDREN, AND ADULTS

J. A. Desor, Owen Maller, and Lawrence S. Greene

There are many compounds critical to human nutrition that are detected in the environment only by the taste system. Two of these are routinely available in highly purified form as odorless, white crystals on tables, counters and wherever else food is consumed in many parts of the world. They have drastically different physiological effects; both are very relevant to nutrition. It is only when they are tasted that one can differentiate between them. Sodium chloride is essential in maintaining electrolyte balance, and sucrose is an excellent source of calories. Their consumption is mediated by the individual's preference for saltiness or sweetness as qualities of the foods he ingests. Merely that sucrose is found so consistently available, both where foods are prepared and at the place of actual ingestion, indicates that increasing the sweetness of foods is currently a widespread practice. The determinants of this behavior have been the focus of the series of studies to be described.

INFANT RESPONSES TO SWEET

One of the issues of interest is whether responses to sweet are genetically determined or acquired. One approach to the issue of whether responses to sweet are inherent has been to study the newborn. Any responses present at birth are likely to be genetically controlled and characteristic of the species as a whole.

Infants between one and three days of age were tested to determine what effects sweetening a fluid has on their consumption of it.[4] Two stimulus changes that vary sweetness were used: both the saccharide (from mildly to very sweet) and its concentration were varied. Two monosaccharides, fructose and glucose, and two disaccharides, sucrose and lactose, were tested. Fructose and sucrose are very sweet sugars whereas glucose and lactose are less sweet ones.[14] The sugars were tested in four concentrations: .05, .10, .20, and .30M. Each of the resulting 16 solutions was offered to a sample of 12 infants, 6 males and 6 females.

The infants were tested approxi-

Acknowledgment: Supported in part by PHS grant #HD 06781–02 from the National Institute of Child Health and Development. W e express our gratitude to the staffs of the Section on Newborn Pediatrics and the Well Baby Clinic of Pennsylvania Hospital for their assistance and cooperation. We are especially appreciative of the enthusiastic and dedicated assistance of Kathryn Andrews and Robyn Resnick.

mately midway between regular feedings. They were offered water for three minutes and a sugar solution for three minutes with a two-minute rest between these presentations. Half of the infants of each sex received water first, and the rest received the sugar solution first. The volumes ingested during these controlled periods were measured.

The newborns discriminated between plain and sweetened water. They consumed more fluid when it was sweetened. Further, they were responsive to the degree of sweetness. When solutions were made sweeter by increasing concentration, the infants responded by taking proportionately more of the solution (Figure 12–1). They were also responsive to the differing sweetness of the sugars used (Figure 12–2). Sucrose and fructose were more effective in increasing the volume consumed than were lactose and glucose.

This increased consumption in response to sweet was observed using a water medium.[4] It has also been found when sweetness was varied in a milk medium.[15] It is quite clear that humans are responsive to sweet soon after birth, thus suggesting that the response is innate. The type of responsiveness observed in these studies is one affecting intake. Over the sweetness levels tested, as a fluid is made sweeter, greater quantities of it are taken into the gastro-intestinal tract.

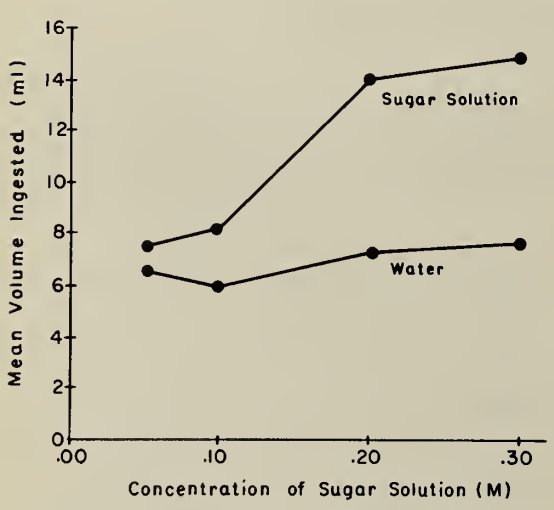


Figure 12–1. Mean volume of sugar solutions and water ingested by infants offered different concentrations of sugar solutions. Copyright 1973 by the American Psychological Association. Reprinted[4] by permission.

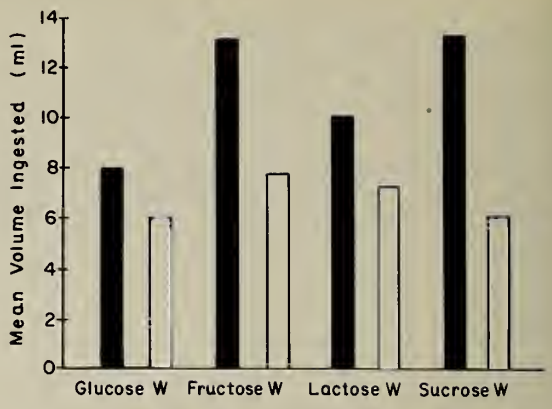


Figure 12–2. Mean volume of sugar solutions and water (W) ingested by infants offered solutions of different sugars. Copyright 1973 by the American Psychological Association. Reprinted[4] by permission.

Effect of other taste qualities on the response to sweet

The facilitation of intake in the newborn by sweet appears to be unaffected by the addition of salty or bitter tastes. Sour stimuli, however, suppress the effect. In testing for newborn responses to tastes other than sweet, the testing situation described above was employed to determine the effectiveness of sweet in mixture with other taste qualities.[3] The high level of consumption of sweet fluids was unaffected by the addition of urea in

concentrations as high as .48 M (Figure 12–3) or salt in amounts up to .2M (Figure 12–4). Adding citric acid in concentrations from .001 to .024 M to the sweet fluid (a .07 M sucrose solution) resulted in a reduction in intake (Figure 12–5).

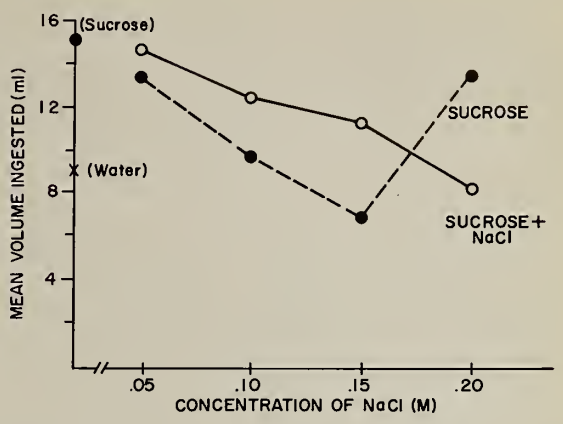


Figure 12–4. Mean volumes of .07 M sucrose with and without NaCl ingested by newborns offered different concentrations of NaCl (n=12).

Preference for sweet relative to milk

It is clear that newborns are not passive receptacles of fluids offered to them. They actively vary the volume they consume, taking more or less depending on the sensory qualities of the fluids offered. Infant formula, like solutions of sweet carbohydrates, is consumed in greater quantities than is water.[11] Sugar solutions and milk formula both provide nutrients that are absent when plain water is offered. Formula, however, is a simulation of the newborn's complete and natural diet. If relative palatability reflects relative nutritive value, then newborns should demonstrate greater preferences for milk formula than for sugar solutions. A comparison of the two was made using the testing technique described previously. A group of 15 infants was offered a low density milk formula (Similac 13) and a .3 M sucrose solution for three minutes each. The two fluids were equated for caloric density (.4 Kcal/ml). Sucrose was used since it is selected by adults as highly acceptable among sweet carbohydrates.[1]

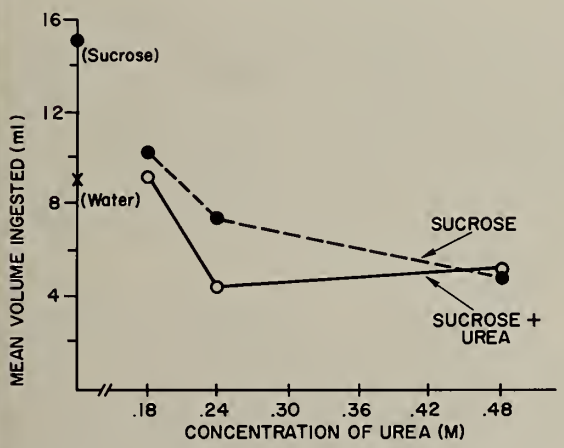


Figure 12–3. Mean volumes of .07 M sucrose with and without urea ingested by newborns offered different concentrations of urea (n=12).

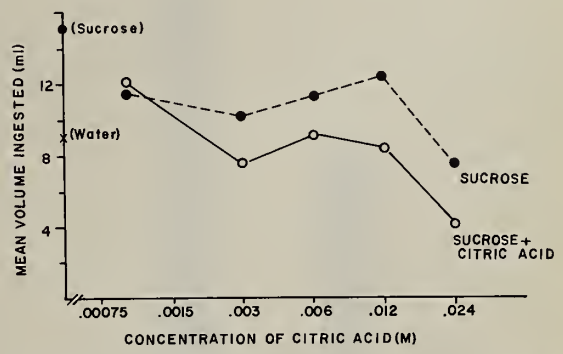


Figure 12–5. Mean volumes of .07 M sucrose with and without citric acid ingested by newborns offered different concentrations of citric acid (n=12). Copyright 1975 by the American Psychological Association. Reprinted [3] by permission.

The newborns demonstrated greater preferences for the sucrose solution than for the milk formula (t = 2.8, df = 14, p. <.05). The mean volumes ingested of each during the three-minute periods are given in Figure 12–6. Three of the subjects consumed the maximum volume offered of the sucrose solution (40 ml) in less than the three-minute period. Presumably, the differential intake of the two fluids would have been even more pronounced had there been no limits on the volumes the infants were allowed to ingest during the test. Within this restricted situation, the newborns consumed over twice the number of calories when the caloric source was the sucrose solution rather than the milk formula. This preference for the sweet carbohydrate relative to milk was observed in a short-term test where postingestional factors were not likely to be of consequence. Thus, the preference is presumably a sensory preference, an immediate hedonic response to the perceptual qualities of the fluids, that describes their relative palatability. This short-term preference may not reflect long-term intake preferences when postingestional effects are operative.

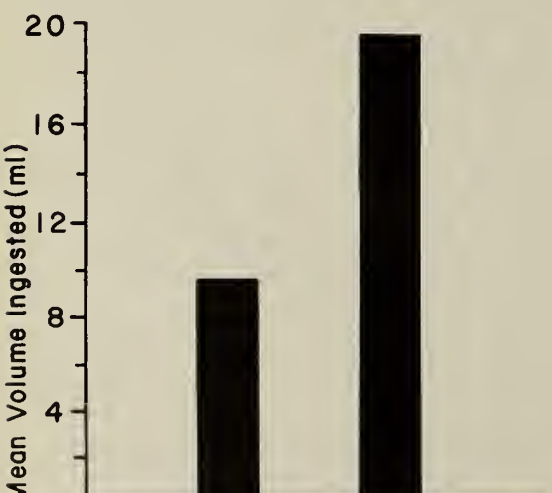


Figure 12–6. Mean volumes of milk formula and an isocaloric sucrose solution ingested by human newborns (n=15).

Effect of hunger/thirst on sweet preferences

All of the tests with newborns described thus far were conducted approximately midway between regularly scheduled hospital feedings. These feedings occurred approximately every four hours. Presumably, the infants were neither as hungry nor as satiated as their feeding regime allows. It was of interest to determine whether the preferences observed are specific to this intermediate level of hunger and thirst or are generally stable over levels of deprivation.

Tests as were described above were repeated for three sugar solutions, .2 and .3 M glucose and .2 M fructose. In this study,[12] each infant was tested not once, but twice, under different levels of deprivation, and one-minute rather than three-minute ingestion periods were used. One test preceded a regularly scheduled feeding, ending two minutes before the feeding. The other test followed a regular feeding, beginning two minutes after the completion of the meal.

The higher concentration of glucose was a more effective stimulus in increasing ingestion than was the lower, and this was independent of deprivation. Fructose was more effective than the isomolar solution of glucose in stimulating consumption in the four-hour deprived infants, but not in recently fed infants.

Greater quantities of all the stimuli, water and sugar solutions alike, were consumed when the infants were tested before a feeding rather than after. Hunger and thirst had a generalized effect on the volume consumed by the newborn. Ernits and Corbit [6] found a similar effect in rats. They found that water deprivation resulted in an increase in the volumes of sugar solutions consumed without a substantial alteration of the shape of the preference curve for the solutions.

A similar effect of hunger on preference was observed in human adults. Hill [8] measured the amounts ingested by adults during regular meals. He varied both the level of hunger and relative preference of the meals offered. Increased hunger and increased preference had similar and independent effects: both resulted in an increase in the amount consumed. It appears that newborn and adult humans vary consumption in response to both deprivation and the sensory qualities of the foods offered.

Responses of older infants

It is of interest to determine whether the response to sweet present in the newborn persists or is subsequently modified developmentally and/or as a result of dietary experience. Two groups of older infants were tested with the same procedure used in studying newborns. One group ranged from 5 to 11 weeks of age. These infants had had a considerably longer feeding history than the 1- to 3-day old infants. According to their mothers' reports, milk formula was still the major food, but most had recently been introduced to a variety of other complex foods (e.g., fruit juices, pureed vegetables). An older group, ranging from 20 to 28 weeks of age, were still young enough to be tested by offering nursing bottles, but were on highly varied diets.

In the older infants, fructose and glucose were used to test for responsiveness to differences between sugars. Both .1 and .2 M solutions of each were used to test for a concentration effect. The infants were tested during routine visits to the Well Baby Clinic of Pennsylvania Hospital, and were included as subjects only if the pediatric staff had judged them to be normal and healthy. Their food intake prior to testing was not controlled, and the level of hunger undoubtedly varied somewhat.

Each of the four sugar solutions was offered to 12 infants in each age group. The differences between the volumes of water and test solution ingested were analyzed. The corresponding data from newborns tested with these four sugar solutions [11] were included in the analyses. There were no differences between the three age groups. All demonstrated greater preferences for the .2 M solutions than they did for the .1 M solutions ($F = 11.8$, $df = 1/132$, $p < .01$; Figure 12–7). In addition, fructose was more effective than glucose in facilitating ingestion in all three age groups ($F = 17.4$, $df = 1/132$, $p < .01$; Figure 12–8). Although the degree to which the infants increased their intake in response to sweet did not change over the first six months, the total volume consumed during the six minutes of testing increased with age ($F = 5.6$, $df = 2/138$, $p < .01$).

Newborn and adult comparison

The disposition to allow more of a

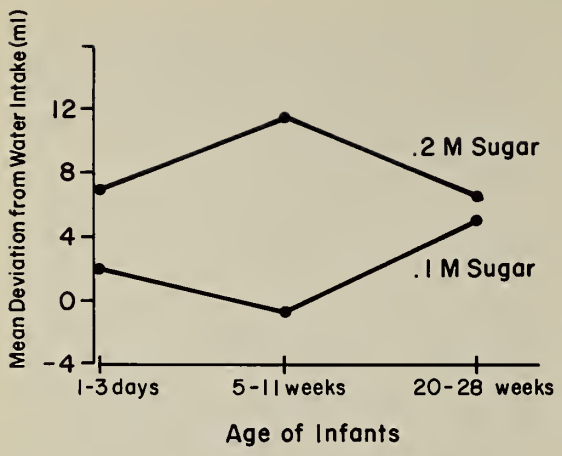


Figure 12–7. Mean differences (n=24) between the volume of sugar solutions and the volume of water ingested by infants differing in age for 2 solution concentrations.

fluid to pass through the oral cavity if that fluid tastes sweet appears to be a characteristic of humans through infancy. It is unaltered from birth through the pre-weaning transition to a varied, omnivorous diet. Is it characteristic throughout the life span? Stellar [18] has presented data on this effect in the adult human. The testing situation [9,18] was remarkably similar to that used here with infants. The subjects were offered a fluid for a 10-minute period. The fluid was delivered through a straw, and the subject could ingest as much or as little as he wanted. Water and solutions of sucrose and glucose, each in seven concentrations were offered. The volume ingested of each of these fluids was measured. Stellar's graphically presented data (Figure 23) were converted to the units used here and are presented along with the corresponding data from newborns in Figures 12–9 and 10. The responses of newborn and adult humans are strikingly similar. It appears that although adults have had extensively varied dietary experiences, they continue to exhibit the same ingestive response to sweet they had at birth.

The adults were tested with higher concentrations of both sucrose and glucose than had been offered to infants. When the higher concentrations are included, a preference-aversion function becomes apparent. This same type of function is charac-

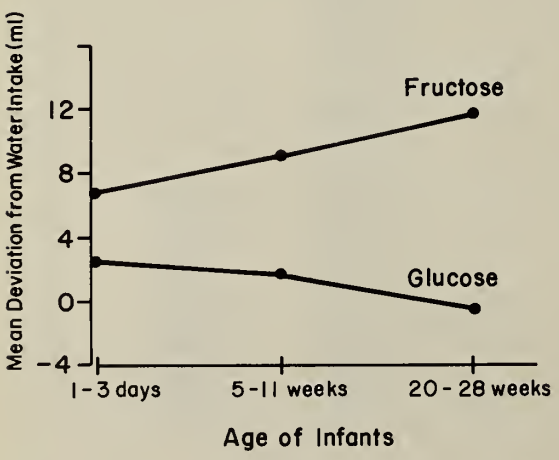


Figure 12–8. Mean differences (n=24) between the volumes of sugar solutions and the volume of water ingested by infants differing in age for 2 sugars.

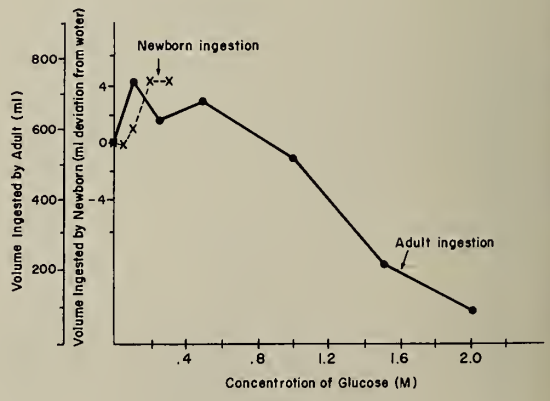


Figure 12–9. Consumption of water and glucose solutions of varying concentration by newborn and adult humans. The newborn data are from Maller and Desor [11] (Figure 18–3) and the adult data are from Stellar [18] (Figure 23).

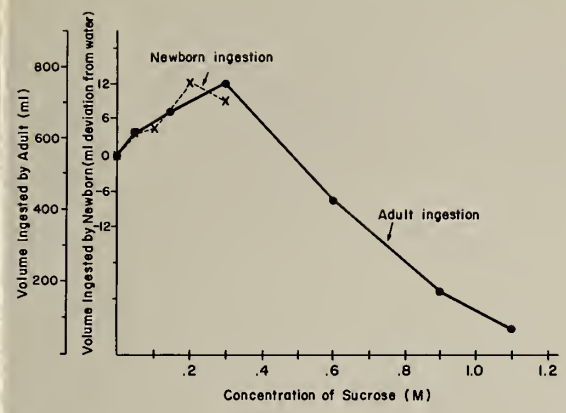


Figure 12–10. Consumption of water and sucrose solutions of varying concentrations by newborn and adult humans. The newborn data are from Maller and Desor [11] (Figure 18–1) and the adult data are from Stellar [18] (Figure 23).

teristic of other species as well.[18] Whether or not the newborn human responds with relative aversion to higher sweetness levels is unknown, although it would be reasonable to expect that he does.

VARIABILITY IN PREFERENCES FOR SWEET

The studies described thus far suggest that a preference for sweet at some intensities is characteristic of the human species. Within the species, however, there are large individual differences in the level of sweetness preferred. The preference-aversion functions given in Figures 12–9 and 10 are typical for averaged responses, but the functions for individuals included in that average vary widely.[5,16] In addition to asking whether there is a heritable *species* preference, one can ask whether individual differences *within* the species are genetically determined. It is possible that a general disposition for sweet is innate whereas the particular intensity of sweetness preferred is determined by experiential, metabolic, and/or situational factors.

To assess the degree to which taste preferences are heritable, a study of twins' preferences was undertaken.[7] Preference for sweet relative to nonsweet was not at issue. These tests were addressed to asking which *level* of sweetness is preferred; i.e., in what concentration does a subject most like to have his sweetness. A group of 311 pairs of twins, 146 monozygotic and 165 like-sex dizygotic, between the ages of 9 and 15 were tested. These samples had approximately equal numbers of males and females and Black and Caucasian subjects.

Each subject was given taste preference tests for three compounds: sucrose, a sweet disaccharide; lactose, a less sweet disaccharide; and sodium chloride. For each of the three tests, the subject was presented with four cups containing different concentrations of the compound. He was asked to taste them without swallowing and indicate which he liked the best, second best, third best, and least. The concentrations offered were .075, .15, .30, .60 M sucrose; .10, .20, .30, .40 M lactose; .05, .10, .20, .40 M NaCl. The order in which the concentrations were sampled varied across subjects. The three tests were ordered sucrose, lactose, sodium chloride. They were

separated by approximately one minute and each was preceded by thorough oral rinsing.

Each subject's ordering of the concentrations by preference was transformed into a single preference index, and these indices were used in the analyses. Estimates of heritability, obtained by comparing the correlations between twins' preferences among monozygotic and dizygotic pairs, were calculated for all three compounds. There was no evidence that the level of sweetness or saltiness preferred by the individual is under genetic control. The correlations between twins' preferences were uniformly low in both the monozygotic and dizygotic groups. Thus it appears that having the same genetic material and/or being raised in the same household does not imply similarity of taste preferences.

Sex and race differences in sweet preferences

In this study of heritability of preferred levels of sweetness and saltiness, a very large sample of adolescents was tested. The distribution of preference levels for sucrose among these 9 to 15 year old subjects was skewed, with greater numbers of them indicating that they prefer sweetness in the higher concentrations. The same was found with lactose, although it was much less pronounced. The opposite tendency was observed with sodium chloride: higher proportions of subjects indicated they prefer saltiness at low intensities.

Within this sample of 9 to 15 year old subjects, the males demonstrated preferences for higher levels of sweetness than did the females. There was no sex difference in the distributions of levels of saltiness preferred. In addition, the Black and Caucasian subjects differed in their taste preferences. For all three compounds, the Blacks indicated higher preference levels than did the Caucasians. The distributions of preference for sucrose in the two racial groups are given in Figure 12–11. The preference for higher intensities of sucrose among Blacks relative to Caucasians was found in both high and low socio-economic groups and for both sexes.

Adolescent and adult comparisons

The subjects in the twin study ranged from 9 to 15 years of age. There were no changes in the distribution of preferences over this range of ages spanning the onset of puberty. An additional study was undertaken to determine whether the distribution of preferences observed in these children and adolescents is also characteristic of the mature adult population.[2]

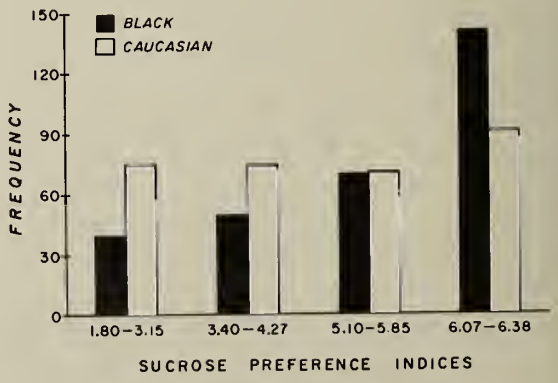


Figure 12–11. Frequency distributions of cumulative sucrose taste indices by race. Copyright 1974 by the American Psychological Association. Reprinted [7] by permission.

Preference tests were given to 140 adults ranging in age from 18 to 64 years. There were 92 males and 48 females, 52 Blacks and 88 Caucasians in this group. The same materials and procedure described above were used with this adult sample.

The distributions of first-choices for the 9–15 year old and adult groups were compared. For each of the three compounds, more of the younger subjects selected higher concentrations as their most preferred. The proportions of 9–15 year and adult subjects selecting each of the concentrations as the one they like the best are given in Figure 12–12. For sucrose, the adults were approximately evenly distributed among the four sweetness levels whereas among the younger subjects, approximately half of them most preferred the highest sweetness level offered. This effect was replicated with lactose, although it was less pronounced. Interestingly, the same tendency for more of the 9–15 year old subjects to demonstrate preferences for the more intense taste was observed with salt. In both groups, the large majority of subjects indicated that they most prefer saltiness in low concentrations. In the younger group, however, there was a large group of Blacks (30%) preferring the highest level of saltiness. Preferences for salt among the 9–15 year old Caucasians were distributed like those of the adults.

Within the adult population, neither racial nor sex differences were observed. In addition, no further age differences were observed among the adults. The 56 subjects between 18 and 29 years of age were compared to the 48 oldest subjects, ranging from 45 to 64 years of age. These two age groups did not differ in their taste preferences for any of the three compounds tested.

Comparison of ingestive and psychophysical measures of preference

These studies of within-species variation make it clear that there are indeed differences between individuals

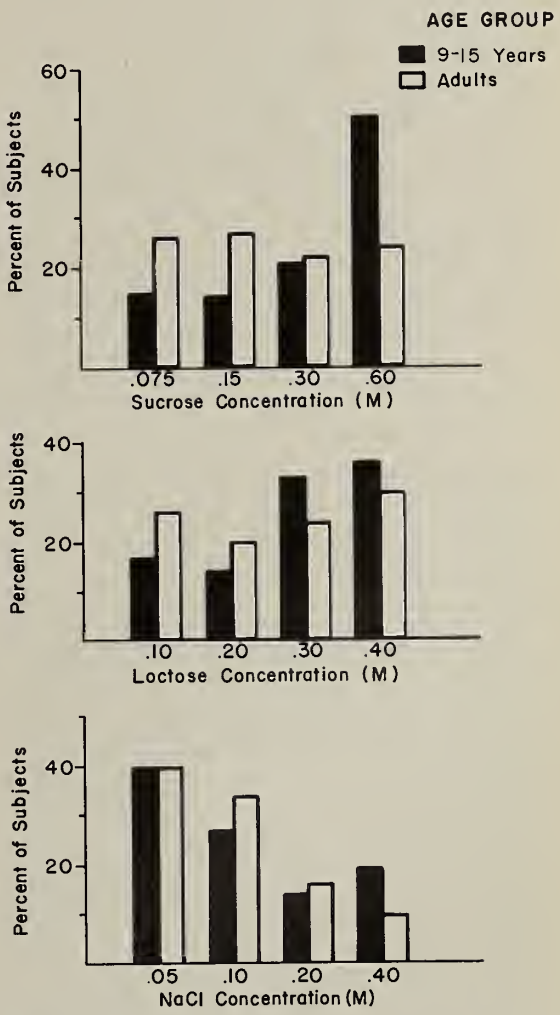


Figure 12–12. Percentage of adolescents and adults selecting varying concentrations of sucrose (upper figure), lactose (middle figure), and sodium chloride (lower figure) as their most preferred concentration.

in the level of sweetness they most prefer. The population is not homogeneous, and the data collected here suggest large numbers of people differ by as much as eight-fold in their preferred level. The preference-aversion function for ingestion of sucrose by adults (Figure 12–10) shows that the concentration of maximum preference is approximately .3 M. About one-fourth of the adult population most prefers sucrose at approximately this level (Figure 12–12). Another one-quarter of the adults expressed a maximum preference for double this sweetness level, and fully one-half indicated they most prefer their sucrose at much lower concentrations.

The preference test used here can be viewed as having asked subjects to specify parameters of their preference-aversion function. The first choice should indicate approximately where the peak of their curve falls. If the test is a valid measure of ingestive behavior, then the ingestive maximum given in Figure 12–10 should be calculable form the first-choice data given in Figure 12–12. The mean concentration of maximum preference for the 140 adults tested here is .27 M, a value in excellent agreement with the peak of the averaged ingestive data collected by Stellar and his colleagues.[18] If a group of subjects between 9 and 15 years of age were tested in the feeding situation used by Jordan, Weiland, Zebley, Stellar, and Stunkard,[9] their ingestive preference curve should reach a maximum at a higher concentration than did the curve for adults. The mean concentration of maximum preference for the 608 9–15 year old subjects described in Figure 12–12 is .39 M. Further, the preference data predict that the ingestive function for Black children and adolescents will peak at a higher concentration than that for Caucasians of the same age, and males and females will differ similarly.

Stability of individual preferences

The preferences for sweet described above summarize single measurements on large groups of subjects. It is critical to know whether the preference demonstrated by a subject on this test is transitory or stable. It is possible that an individual's preference for sweet varies over time, perhaps with the social situation and/or his metabolic state. If, for example, preference for sweet correlates with hunger, then the differences observed between the two age groups may reflect only that at any given time, the level of hunger among those 9–15 years of age is greater than that among adults. Alternatively, the preference level for sweet may be a fairly stable characteristic of the individual, determining more generally the level of sweetness he selects.

In order to measure the stability of these preferences as well as the reliability of the test, 27 of the adults were retested approximately two months after their first test.[7] The preference indices reflecting the ordering of the four concentrations on the test were calculated. The test-retest correlations (r) for these were .72 for sucrose ($p<.001$), .54 for lactose ($p<.01$), and .88 for sodium chloride ($p<.001$). In order to observe such correlations, there must be both a stable characteristic to measure and a reliable method of measuring it. The

degree to which these preferences were not constant, of course, reflects variability in the preference system, the measurement technique, or both.

It is clear that there is some individual consistency in the level of sucrose most preferred. In addition to any immediate factors that may determine preference, there are stable preferences characteristic of the individual. That is, it appears that some individuals can be characterized as liking strong concentrations of sucrose best and others are characterizable as most preferring it in low concentrations.

It does not necessarily follow that because some parts of the population prefer only mild sweetness, they consume fewer calories in sweet nutrients. It is possible that an individual's taste preference level mediates the amount (or proportion) of sucrose in his diet. Alternatively, this preference may determine the *form* in which sucrose is ingested without being related to total sucrose consumption. For example, an individual preferring concentrated sweetness may eat candy when he eats sugar at all and ingest only small amounts of candy. Another, preferring mild sweetness, may take small amounts of sucrose in nearly everything he ingests, adding it in low concentrations to coffee or tea and routinely selecting mildly sweetened vegetables, meats, etc. (e.g., cole slaw, stewed tomatoes, barbecued meats). In the absence of research correlating preferences with long-term intake patterns, it is premature to suggest that the observed differences in preferences imply dietary and/or nutritional differences.

SUMMARY

The studies summarized above suggest the following is true of humans with regard to sweet: there is a preference for sweet at some levels relative to nonsweet, it is an unlearned preference present from birth, and it appears to be unaltered from birth through adulthood. Members of the species are not homogeneous in the level of sweetness they prefer, and the individual's preferred level appears to be a fairly stable characteristic of him. There was no evidence that individual differences in preference level are due to genetic or immediate familial factors. Various subgroups differ in the proportions of individuals having preferences for low, medium, or high sweetness. In a westernized, urban population, such differences were observed between groups differing in age, sex, and race.

Sweet appears to be a fairly unique sensory quality. To find a species-wide preferential response to one of four presumed primary taste qualities is analogous to finding a genetic preference for red, green, or a particular pitch. It is peculiar to find that a single sensory quality, rather than mediating differentiation between whole percepts, is reinforcing in and of itself. This inherent affective response to the sensory quality of sweet is not unique to humans. Sweet is a primary reinforcer to many species, including many mammals, [10,13,17] and from phylogenetic inference alone, the same would be expected in humans.

It is not surprising, therefore, that sucrose, being of highly acceptable sweetness, is found so pervasively

where humans prepare their foods. In view of the wide individual differences in the level of sucrose preferred, it is equally unsurprising that this compound is present at the place of actual ingestion, thereby allowing the individual to determine the level he ingests.

REFERENCES

1. Chappell, G.: Flavor assessment of sugar solutions. *J. Sci. Food Agric.*, 4: 346–350, 1953.
2. Desor, J., Greene, L., and Maller, O.: Preferences for sweet and salty in 9- to 15-year old and adult humans. *Science*, 190:686–687, 1975.
3. Desor, J., Maller, O., and Andrews, K.: Ingestive responses of human newborns to salty, sour, and bitter stimuli. *J. Comp. Physiol. Psychol., in press.*
4. Desor, J., Maller, O., and Turner, R.: Taste in acceptance of sugars by human infants. *J. Comp. Physiol. Psychol.*, 84:496–501, 1973.
5. Ekman, G. and Akesson, C.: Saltness, sweetness, and preference. *Scand. J. Psych.*, 6: 241–253, 1965.
6. Ernits, T. and Corbit, J.: Taste as a dipsogenic stimulus. *J. Comp. Physiol. Psychol.*, 83: 27–31, 1973.
7. Greene, L., Desor, J., and Maller, O.: Heredity and experience: Their relative importance in the development of taste preference in man. *J. Comp. Physiol. Psychol.*, 89:279–284, 1975.
8. Hill, S.: Eating responses of humans during dinner meals. *J. Comp. Physiol. Psychol.*, 86: 652–657, 1974.
9. Jordan, H., Weiland, W., Zebley, S., Stellar, E., and Stunkard, A.: The direct measurement of food intake in man: A method for the objective study of eating behavior. *Psychosomatic Med.*, 28: 836–842, 1966.
10. Kare, M. and Ficken, M.: Comparative studies on the sense of taste. In: Zotterman, Y. (Ed.): *Olfaction and Taste.* New York, Pergamon, 1963.
11. Maller, O. and Desor, J.: Effect of taste on ingestion by human newborns. In: Bosma, J. (Ed.): *Fourth Symposium on Oral Sensation and Perception.* Washington, U.S. Govt. Printing Office, 1974.
12. Maller, O. and Desor, J.: Sugar preferences of human newborns as a function of hunger. In preparation.
13. Maller, O. and Hamilton, C.: Sucrose and caloric intake by normal and diabetic monkeys. *J. Comp. Physiol. Psychol.*, 66: 444–449, 1968.
14. Moskowitz, H.: The sweetness and pleasantness of sugars. *Am. J. Psych.*, 84: 387–405, 1971.
15. Nisbett, R. and Gurwitz, S.: Weight, sex, and the eating behavior of human newborns. *J. Comp. Physiol. Psychol.*, 73: 245–253, 1970.
16. Pangborn, R.: Individual variation in affective responses to taste stimuli. *Psychonomic Sc.*, 21: 125–126, 1970.
17. Sheffield, F. and Roby, T.: Reward value of a non-nutritive sweet taste. *J. Comp. Physiol. Psychol.*, 43: 471–481, 1950.
18. Stellar, E.: Hunger in man. *Am. Psychologist*, 22: 105–117, 1967.

Chapter 13

FACIAL EXPRESSIONS OF THE NEONATE INFANT INDICATING THE HEDONICS OF FOOD-RELATED CHEMICAL STIMULI

Jacob E. Steiner

In the very first hours of extra-uterine life the human neonate responds to food related chemical stimuli with facial expressions. I shall try to demonstrate that such neonatal facial expressions can be viewed as non-verbal communication signals that indicate willingness to accept or need to reject a stimulus. The stimulus-induced differential facial expressions of acceptance or aversion indicate the existence of a discriminatory mechanism that appears to provide an evaluation of a hedonic dimension of chemical stimuli related to food. The circumstances of the observations indicate that such a "hedonic monitor" operates on a non-cognitive level and that its evaluation of stimuli is not directly or primarily related to the previous experience of the organism or to a learning process. The study of the development of sweet preference may thus be approached under the assumption that man, like many other organisms, has a "natural" preference for sweet.

Kalmus, in a paper presented at the IV. International Symposium on Olfaction and Taste,[13] states a position germane to our concern with response to food related stimuli.

> My proposition is that it will be profitable to investigate the more integrative faculties of chemoreception. Such studies must start from considerations of the general role of chemical perception in the life of animals and from there proceed to a detailed analysis of the tasks which the gustatory and olfactory senses must perform in the maintenance of life . . . Belief in the faculties of our nose and tongue to discover what is wholesome and what is deleterious is wide spread.

There is little doubt that the two major chemical senses, gustation and olfaction, play an important role in the search for and selection of food throughout the Animal Kingdom. Man is considered to be microsmatic, i.e. to have limited ability to utilize olfactory cues. Yet, even he makes good use of olfactory signals to distinguish fresh from rotten food, and as basis for his food related feeling of enjoyment or disgust. Gustatory cues can be decisive in motivating the behavior of men and animals. As Pfaffmann demonstrated, the clear correlation between behavioral and electro-physiological measures support the assumption that gustatory stimuli are involved in the control of consumatory responses and the re-

inforcement of instrumental behavior.[17] Recent observations including our own, on human chewing habits, give additional support to Pfaffmann's view.[14,22] In his paper dealing with "The Pleasures of Sensation," Pfaffmann observes "that there is increasing support for the idea that gustatory stimulation *per se* is capable of eliciting and reinforcing behavior in its own right:—We might say 'sweet for sweet's sake.' " [17]

THE GUSTOFACIAL RESPONSE

In the course of psychophysical testing we became aware, that suprathreshold taste stimuli in the sweet, sour and bitter submodalities of taste elicited certain "grimaces" from adults. The facial expressions observed in a diverse adult population were surprising in many respects. The facial expressions elicited by the three taste qualities were different from each other, and also different from the response elicited by similar stimulation with distilled water. The responses were impressively consistent, and of very short latency. These preliminary observations which were in fact a by-product of other studies,[8,9,18,25,26] suggested that these reactions may be primarily innate rather than acquired. Therefore, a systematic study of the neonate was undertaken.

Method

There are three groups of subjects. One group consists of 100 termborn normal neonates of both sexes, tested between 3 and 7 days of age; a second group includes 75 termborn normal neonates of both sexes tested before the first breast (or bottle) feeding. Four neonates, born with severe developmental malformations of the CNS (2 of them cases of Anencephaly, 2 of them cases of Hydro-Anencephaly) comprise the final group. All neonates are stimulated with the same tastants, one for each of three taste-modalities. The stimuli elicit clear reports of sweet, sour or bitter taste from normal adult humans. The concentrations of the aqueous solutions are as follows: 25% sucrose for sweet, 2.5% citric acid for sour, and 0.25% quinine sulphate for bitter.

The neonates receive the stimuli at room temperature from disposable sterile plastic pipetts. For each stimulation 0.5 ml of liquid is rinsed on the dorsal surface of the tongue. Stimuli are presented to the infants in a random sequence. Records of the elicited facial expressions are made by cinema, videotape or still-photography. No additional lighting beyond that normally present in the newborn nurseries is employed. For purposes of comparison the resting face (without any stimulation) of each infant is recorded, as well as the face after presentation of distilled water. The recordings are evaluated by a panel of observers who did not know the nature of the stimuli. Only responses unaccompanied by vocalization are analyzed.

Results

The facial expressions can be separately characterized according to the stimuli that elicit them. The sweet stimulus leads to a marked relaxation of the face, resembling an expression

of "satisfaction." This expression is often accompanied by a slight smile and was almost always followed by an eager licking of the upper lip, and sucking movements. This licking and sucking is almost "aloud" sucking. The facial play elicited by the sucrose stimulus was labeled by the observers of the films and pictures as an expression of appreciation, liking or enjoyment. The sour stimulus leads to a lip-pursing, in a fashion known as the "Darwin's pursing of the lips," may be either continuous or repetative. This is often accompanied or followed by a wrinkling of the nose and blinking of the eyes. Stimulation with the bitter fluid leads to a typical arch form opening of the mouth with the upper lip elevated, the mouth angles depressed, and the tongue protruded in a flat position. This expression involves primarily the mouth region of the face and includes the elements typical of a facial play expressing dislike and disgust or rejection. It was often followed by spitting or even by the preparatory movements of vomiting.

The resemblance of these neonatal expressions to those observed in the adult is impressive. The facial expressions are displayed by the preponderant majority of the normal termborn neonates and by all four neonates with the developmental malformations of the CNS.

Figures 13–1 and 2 give a synoptic view of the typical facial expressions as observed both in the normal neonate and the neonates with developmental malformations of the CNS. Table 13–I summarizes the frequency with which the main components of facial expression occur as a response to each of the three gustatory stimuli. Since the analysis is limited to responses which are not accompanied or followed by any vocalization whatsoever, the tables, the figures and the summary remarks below all relate to exclusively non-verbal, non-vocal motor reactions.

Summary

Sweet, sour and bitter stimuli elicit differential and typical facial expressions in the neonate infants. Since the neonate can display differential facial expressions to various tastants in the very first hours of its extrauterine life, prior to any feeding experience, it can be assumed that the discriminative facial expressions are present at birth and not acquired by life-experience and learning. From the fact that neonates without brain structures other than the brain stem and mesencephalon can react to tastants in a fashion similar to normal termborn neonates, one can conclude that the gustofacial response is a low-level reflex-like response, that does not involve cortical structures. A neural pathway consistent with these findings has been proposed elsewhere.[19]

Adult observers, viewing pictures of these expressions without knowledge of the stimulus applied interpret the facial expressions elicited by sweet in terms of satisfaction, appreciation, enjoyment or liking, and the responses induced by bitter in terms of disgust, aversion, or disliking. Thus these facial expressions of neonates have clear communicational value for adults. These signals are to be evaluated as a part of the catalogue of non-verbal communicational patterns broadcasted by the infant to his mother.

Figure 13–1. Typical features of the Gustofacial Response, recorded in neonate infants between birth and the first feeding. 1: Resting face. 2: Reaction to distilled water (control). 3: Response to sweet stimulus. 4: Lip-pursing, response to sour stimulus. 5: Response to bitter stimulus.

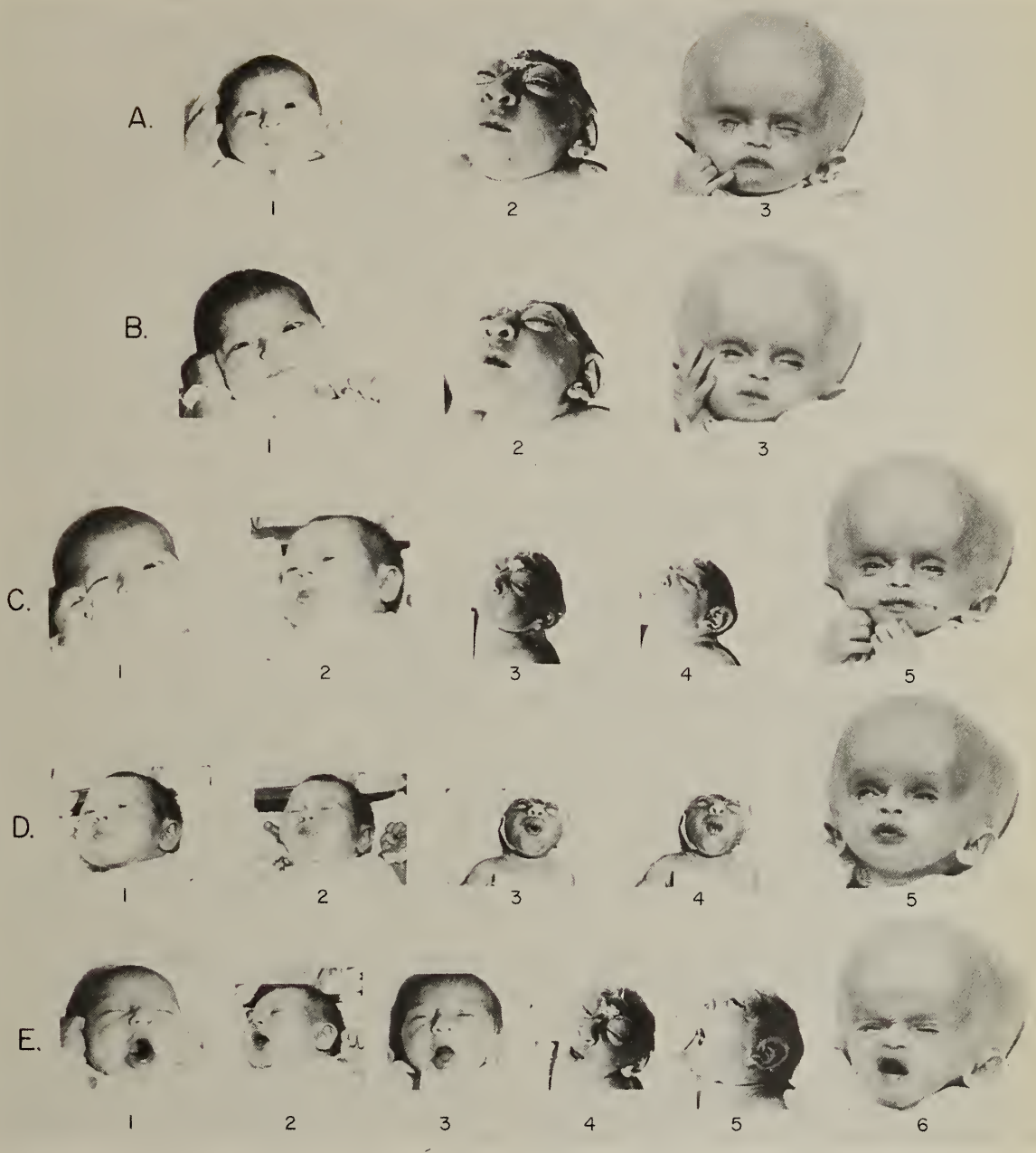


Figure 13–2. Synopsis of the facial response to tastants in normal, anencephalic and hydro-anencephalic neonates.

A) Resting face (without stimulation). 1=normal, 2=anencephalus, 3=hydro-anencephalus.
B) Control face (dist. water stimulation). 1=norm. 2=anenceph. 3=hy. anenceph.
C) Sweet-response.1,2 = norm. 3,4 = anenceph. 5 = hy. anenceph.
D) Sour-response.1,2 = norm. 3,4 = anenceph. 5= hy. anenceph.
E) Bitter-response.1,2,3 = norm. 4,5 = anenceph. 6 = hy. anenceph.

TABLE 13–I

THE MOST CHARACTERISTIC FEATURES OF FACIAL RESPONSE TO GUSTATORY STIMULI AMONG NORMAL AND ANENCEPHALIC NEONATES

Feature	*Percentage of termborn normal neonates*		*Number of anencephalic neonates*
	below 20 hrs. n=75	*7–8 days n=100*	*1–19 days n=4*
		SWEET	
Retraction of mouth angles	81	87	4
Clear expression of satisfaction resembling "smile"	77	73	3
Eager sucking with licking of the upper lip	99	97	4
		SOUR	
Pursing of lips	100	98	4
Wrinkled nose	77	73	2
Quick repeated blinking	89	70	2
Increased salivation	81	65	4
Flushing	76	64	1
		BITTER	
"Arch-like" lips with depression of mouth angles	97	96	4
Protrusion of flat tongue	79	81	3
Salivation and spitting	76	87	4
General expression of "anger" and "dislike"	79	86	3
Vomiting	45	52	3

FACIAL EXPRESSIONS INDUCED BY FOOD ODORS

A second investigation explores the possibility that food related odors might also elicit discriminative facial expressions from the neonate. In this context it is perhaps useful to remember that elements very similar to those elicited by pungent odors are contained in the grimace and facial play that man, even civilized man, employs to express aversion or dislike.

This study [23,24] was carried out in Philadelphia in collaboration with Dr. Loretta Finnegan, director of the Newborn Nurseries at the Philadelphia General Hospital.* We finished this study several days prior to this symposium. A preliminary note on these observations is included in a dis-

* This study was carried out while the author was on a sabbatical leave from his home institution, The Hebrew University Hadassh School of Dental Medicine, Jerusalem, and was appointed as Visiting Associate Professor at the University of Pennsylvania School of Dental Medicine.

cussion paper presented on another occasion.[20]

Subjects

The subjects are twenty-five termborn normal Caucasian and Negro neonates of both sexes. Each subject is tested between birth and first feeding. Records are made of the resting or unstimulated face and of the response to a cotton swab dipped in the odorless solvent used to dilute the test odorants. Still and motion-picture photographs are taken under available light as in the previous study.

Stimuli

Odor stimuli sample the wide spectrum of odors, related to acceptable and unacceptable nutrients. Since real foodstuffs could not be introduced into the nursery the stimuli are artificial food flavors, approved by the U.S. Food and Drug Administration.*

A vanilla and a banana represent the "fruity" type of food odors while artificial butter flavor represents the "milky" odors. Food-related odors of negative hedonic value are a concentrated artificial shrimp flavor, which smelled "fishy," and a non-toxic flavor of rotten eggs that represent the smell of rotten food. Hedonic judgements of these odors made by 10 student volunteers on a 9 category scale show almost unanimous agreement as to the pleasantness of the banana, vanilla and butter odors. The rotten egg flavor is clearly aversive, but the fishy odor is not as clearly offensive.

The photographs of the infants are evaluated by a panel of three medically trained and three naive judges. The facial expression photographs, coded by number, are judged in the following terms: 1) acceptance (baby likes it, enjoys it), 2) aversion (baby dislikes it, it causes him disgust), 3) indifference (judge cannot discriminate the particular reaction from the resting face control). Table 13–II gives the coefficients of agreement and the distribution of judgments among the categories when responses of 25 infants to each of five odor stimuli were judged.

Findings and Conclusion

Normal termborn neonates of both races tested display differential facial expressions to food related odor stimuli at birth, prior to any feeding experience whatsoever. This speaks for the possibility that the phenomenon is a general human reaction (see Figure 13–3).

The photographically documented facial expressions can be easily interpreted by observers and classified as expressions of liking, enjoyment, satisfaction or of dislike, disgust, aversion, respectively. There appears to be valid communicational aspects of the observed expressions. One may assume that if an objective outsider is able to clearly interpret the signals emitted by the neonate, such signals would be even more effective for the infant's own mother.

Facial expressions evaluated by the observers as those of acceptance are elicited by flavors, that adults find pleasant. Those facial expressions which are evaluated as reflecting aversion are induced by stimuli which

* We wish to express our appreciation to Firmanich Inc. N.Y., for preparing and supplying the flavors used as stimulants in this study.

Figure 13–3a. Facial expressions of infants in response to odors, which are pleasant to adults.

Figure 13–3b. Facial expressions of infants in response to odors, which are unpleasant to adults.

TABLE 13–II

JUDGEMENTS OF THE FACIAL RESPONSES OF 25 NEONATES BY 6 JUDGES

ODOR STIMULUS	*Agreement coeff* [(a)]	*Distribution of judgements in %*		
		Acceptance [(b)]	*Indifference*	*Rejection*
Butter	0.59	62	32	6
Banana	0.35	55	25	20
Vanilla	0.63	46	46	8
Shrimp (Fishy)	0.23	22	25	53
Rotten eggs	1.00	—	—	100

Notes:
(a) For each stimulus the agreement coefficient is a mean of the 15 correlation coefficients derived by correlating the judgements of each of 6 judges with every other judge.
(b) See text for description of categories

adults find pungent or disgusting. (See Figure 13–4).

Even on the basis of the less than perfect imitation food flavors of this study, neonates discriminate odors typical of fresh and valuable nutriants, from the odors of food which the adult would "instinctively" reject. A hedonic monitor system is implied.

Since the reaction can be observed at birth and such subjects could not have had any previous experience, the reaction can be called "innate" or "inborn."

Evidence for the existence of this discriminative facial play in neonates, born with developmental malformations of the CNS is not available from this series of observations. However, by analogy with the Gustofacial response, we assume that the response to olfactory stimuli is possible without participation of higher brain structures, but at this stage cannot propose what neural pathways are responsible for it.

FURTHER EXPERIMENTS AND OBSERVATIONS

Additional experimental projects directly related to the two studies just described are in progress. I would like to mention them briefly and provide some illustrative results.

Observations on a hydroanencephalic neonate

After completing the study on neonatal response to the food odors, I had the rare opportunity to examine another hydroanencephalic infant.* This 12 day old blind infant provides a further demonstration of the gustofacial response. She was also exposed to some of the odor stimuli used in the previous study, and a good quality imitation honey flavor. The artificial honey odor induced a long lasting

* I wish to express my heartfelt thanks to Dr. G. Szabo, Department of Pediatrics at the Hadassah University Hospital, Jerusalem, for turning my attention to this case of hydro-anencephaly.

Figure 13–4. Facial expressions of 5 neonate infants, all of them below 6 hours of age. C is a response to a control swab dipped in an odorless liquid, BA/VA response to the fruity odor (banana or vanilla), FI. response to the fishy flavor (concentrated artificial shrimp), BU. response to a diluted artificial butter-flavor (representative of milky-odor), R.E. response to the odor of rotten eggs.

impressively "joyful" sucking-licking reaction in this infant. It was rather interesting and impressive to observe the eager sucking that appeared immediately after the very first sniff of this flavor. The reactions of this infant are illustrated in Figure 13–5. Having in mind the importance of "sweetish-flavors" as cues for acceptance (banana, vanilla), one may pay special attention to the pictures illustrating the response to honey-flavor. Thus in at least one case of severe developmental malformation of the CNS, food related odors elicit facial expressions similar to those of the termborn normal infants. Autopsy findings clearly indicate that although this infant's brainstem developed normally there is a complete absence of cortical tissue. Even though the histological evidence is still incomplete, the autopsy results suggest that the site of the "hedonic monitor" responsible for differential facial play to odorants is subcortical.

Observations on adults

Our first observations related to the Gustofacial Response were made on adult subjects. Evaluating these earlier observations, we made the assumption that the Gustofacial Response, like many other reflexes, undergoes a suppression during successive development of the cortical functions and is increasingly inhibited. Considering the communicational aspects of this reflex-like response, one would expect that the reflex might continue to appear under appropriate conditions, where it could serve a communicational purpose.

Adults were offered sandwiches which had been specially prepared with margarine that was either sweetened or salted (up to an aversively high degree).[3] When eating in groups, or under other social conditions provocative of a facial play, the subjects display facial expressions resembling the neonate's. However, the adults selectively inhibit the facial responses and do not display them, when eating alone.

Observations on children having congenital carniofacial malformations

Children suffering from craniofacial malformations may undergo rehabilitative surgical procedures that involve massive reconstruction of the bony skull in general and of the frontal area in particular. Such patients often lose the ability to smell when unavoidable damage to the cribriform plate interrupts the olfactory fila. It is of some practical importance in evaluation of these procedures to document the preoperative ability to smell and taste.

Our tests* of some of these children prior to the rehabilitation surgery [21] suggest that the photographic recording of the taste- and smell-induced facial expressions may present an easy, elegant and reliable way to document the child's reactivity to chemical stimuli. The practical application of such recordings is in two directions: First, it is of clinical importance to test the functional state of all senses prior to a massive surgery of the head area. Second, the precise investigation of all

* I would like to express my heartfelt thanks to Dr. Sylvan Stool, Head of the Department of Otolaryngology at the Children's Hospital Philadelphia PA for his collaboration and very kind help.

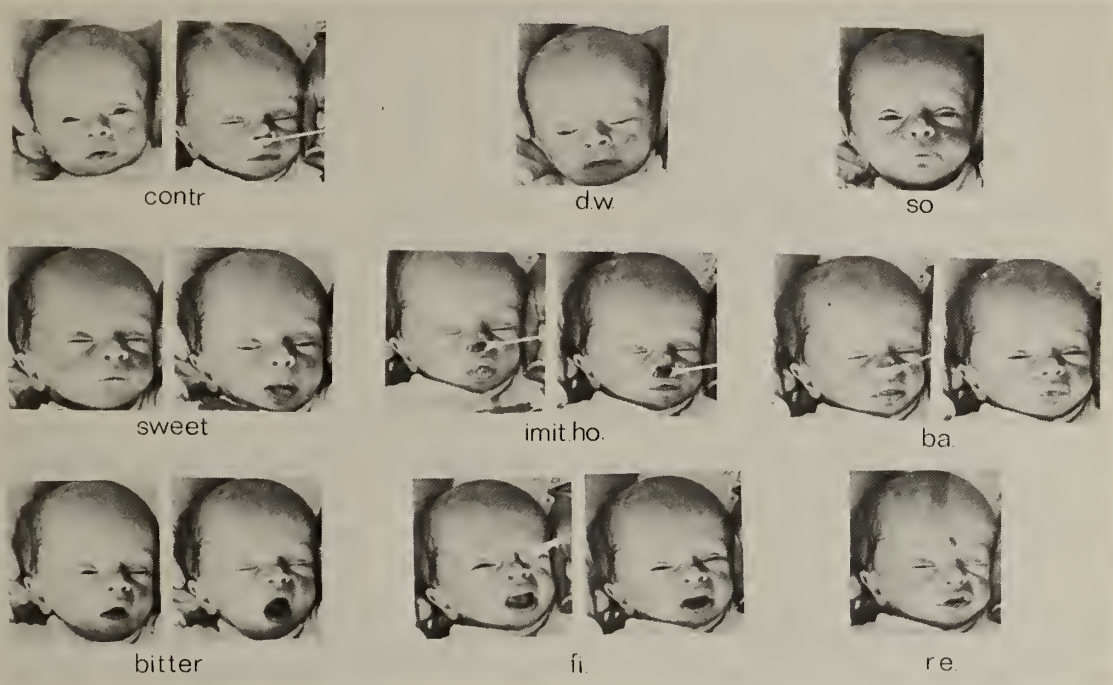


Figure 13–5. Facial expressions of a 12 day old hydro-anencephalic infant to gustatory and olfactory stimuli. *contr* = left frame: without stimulation, right frame: a swab dipped in an odorless liquid. *d.w* = after stimulation with distilled water. *so.* = response to sour stimulus. *sweet* = relaxation and licking as response to sucrose solution. *imit. ho.* a long lasting sucking and licking induced by imitation-honey flavor. *ba.*= response to banana extract. *bitter* = response to bitter tastant. *fi* = aversive response to the fishy odor. *r.e.* = aversive response to the odor of rotten eggs.

sensory and motor functions may provide more accurate dating of the intrauterine event causing the malformation.

Beside these practical applications of the registration and documentation of the facial play induced by tastants and odorants, I think one may draw the following, more general conclusions from these observations:

The human face and its basic expressive play has such profound communicational importance, that even in an extremely distorted human face these basic expressive features maintain their communicational value. Features signalling enjoyment, satisfaction, like, smile; or disappointment, dislike, aversion, fear, respectively, will be easy to read, even from the most distorted human face.

CONCLUDING COMMENTS

The motor reactions described here are stimulus dependent. They are integrated sensory-motor responses of the organism, that can be analyzed and understood from several points of view. I look upon them as part of an innate catalogue of behavioral coordinations, and seek to under-

stand them in terms of human ethology. The evaluation of human facial expression involves many difficulties. The facial expressions of adult man are part of the extremely complex process by which feelings and emotions are communicated. Adults and even adolescents learn to use facial play to express both actual and feigned fluctuations of mood or emotion.

I believe, that a basic catalogue of human facial expressions is innate. From other studies [6,7] we have learned that the smile is neither a learned, nor an imitated facial expression, but rather a communicational tool available even to the congentially blind child. Such children are obviously deprived of the opportunity to mimic facial expressions of the surrounding adults. I used the term innate, but it may be replaced by another one, namely inborn. It is rather difficult to use these terms, without being able to demonstrate that a phenomenon labeled as innate already exists in intrauterine life. For the sake of clear definition I would apply the term innate or inborn to those facial reactions which I can elicit, demonstrate and describe as present at the time of birth. I have good reasons to believe that the basic catalogue of facial expressions are motor coordinations similar to those described and analyzed by the remarkable studies of Hooker [10] and by Humphrey.[11] The expressional tools of the facial play are present at birth, I think their basic form is that which we can observe when these motor coordinations are elicited by a clearly defined isolated sensory stimulus.

Sensory stimuli vary in quality and quantity but they may also have a hedonic dimension. If variation in the qualitative and quantitative aspects of a sensory stimulus can induce differential responding, then the hedonic dimension of a sensory stimulus may lead to a differential response too. I think that hedonics are particularly closely related to an organism's need to send out communicational signals. Such communication might be directed to fellow members of the species, equal in biological rank, or towards members having a special hierarchic ranking or a biological status (e.g. Mother). Sweetness seems to mean acceptability, acceptance of a sweet nutrient can, therefore, lead to satisfaction. In this context I refer to the responses to sweet-substances and also to the "relaxed smiley faces" induced by milky, fruity or honey-like odors.

I would certainly stress that I think that the building-blocks of the basic facial expressive catalogue are available from simple basic circuitary of the brain even in the first phase of life. The gustofacial response may be typical. Based on this assumption, I proposed the neural pathway I sketched in the relevant study.[19] Furthermore, I believe that the same basic circuits, activated in our studies by defined sensory stimuli become available to much more complex coordinations, through learning and other integrations of life experience. The reflection of emotion by means of the same basic facial expressions is possible through involvement of higher brain circuits. I think that the often tragic dissociation between emotions and their motor expressions, which one can witness in chronic mental diseases and in association with injuries, clearly reveal the interrelation

between certain motor processes (such as facial expressions) and emotions.[15,16]

Having analyzed the neonates, responses to attractive and aversive chemical stimuli in terms of the action of a hedonic monitor, one might ask whether this type of stimulus dependent response is uniquely human. I offer some indirect evidence in support of my view that the neonates' responses are a special case of a more general biological phenomenon of motor reactions that reflect hedonic evaluation of chemical stimuli.

In extention of the gustofacial study [19] we carried out animal experiments in which we observed analogies of the human gustofacial response in cats and rabbits. Extensive studies of insects [4,5] have demonstrated delicate motor reactions responsive to the "palatability" of food. Single cell organisms have been known for many decades to actively migrate toward attractive or away from repellent chemical stimuli. This phenomenon has been termed chemotaxis [12] and interest in it is currently undergoing a renaissance. Adler has even demonstrated it in the bacteria *E. Coli.*[1,2] I see the infant's discriminative facial reactions as a very elaborate and complex example of this much more general, perhaps universal, phenomenon. Mentioning Adler's studies, I will conclude with an anecdote. I recently had the opportunity to talk with Julius Adler. In describing our mutual fields of study, he characterized his work as "dealing with smiling and crying bacteria." I think this jocular remark of Adler's may reflect his more serious opinion that, although completely different with respect to techniques and experimental subjects, our studies nonetheless deal with the same basic phenomenon of life.

REFERENCES

1. Adler, J.: Chemotaxis in bacteria. *Science.* 153:708–716, 1966.
2. Adler, J. and Tso, W.: "Decision"-making in bacteria: Chemotactic response of Escherichia coli to conflicting stimuli. *Science.* 184:1292–1294, 1974.
3. Brightman, V.J., Segal, A., Werther, P. and Steiner, J.E.: Ethologic study of facial expressions in response to taste stimuli. *J. Dent. Res.,* 54:A, 1–141, 1975.
4. Dethier, V.G.: The physiology of olfaction in insects. *Ann. N.Y. Ac. Sci.,* 58:139–157, 1954.
5. Dethier, V.G.: The role of olfaction in alcohol ingestion by the blowfly. *J. Insect Physiol.,* 6:222–230, 1961.
6. Eibl-Eibesfeldt, I.: *Grundriss der vergleichenden Verhaltesforschung.* Munchen, Piper Verlag, 1967.
7. Freedman, D.G.: Smiling in blind infants and the issue of innate versus acquired. *J. Child Psychol. Psychiat.,* 5: 171–184, 1964.
8. Halbreich, U. and Steiner, J.E.: Influence of hyperbaric pressure on the sense of taste in man. *Isr. J. Med. Sci.,* 10:5, 1974.
9. Hertz, D., Steiner, J.E., Zuckerman, H. and Pizanti, S.: Psychological and physical symptomformation in menopause. *Psychother. Psychosom.,* 19:47–52, 1971.
10. Hooker, D.: Evidence of prenatal function of the central nervous system in man. James Arthur Lecture on *The Evolution of the Human Brain* for 1957, New York, American Museum of Natural History, 1958.

11. Humphrey, T.: Reflex activity in the oral and facial area of the human fetus. In Bosma, J.F. (Ed.), *Second Symposium on Oral Sensation and Perception,* Springfield, Thomas, 1970, pp. 195–233.
12. Jenning, H.S.: Behavior of the lower organism. 1906. Republished by Indiana Univ. Press. Bloomington, 1962.
13. Kalmus, H.: Minimum central faculties necessary for complex chemical perceptions. In Schneider, D. (Ed.). *Olfaction and Taste IV.* Stuttgart, Wissenschaftliche Verlagsgesellschaft, 1972, pp. 161–166.
14. Pierson, A. and LeMagnen, J.: Etude quantitative du processus de regulation des responses alimentaires chez. l'homme. *Physiology and Behavior,* 4:61–67, 1969.
15. Pilleri, G.: Uber angeborene Verhaltensmuster und deren phylogenetische Wurzeln bei Cortexatrophien. *Psychiat. Clin.,* 2:268–278, 1969.
16. Pilleri, G.: Instinkbewegungen des Menschen in biologischer und neuropathologischer Sicht. *Beitr. z. Verhaltenforschung.* In Blitz, R. and Petrilowitsch, F. (Eds.) Basel, Karger. *Akt. Fragen Psychiat. Neurol.* 11:1–37, 1971.
17. Pfaffmann, C.: The pleasures of sensation. *Psychol. Rev.,* 67:253–268, 1960.
18. Steiner, J.E., Rosenthal-Zifroni, A. and Edelstein, E.: Taste perception in depressive illness. *Isr. Ann. Psychiat. Relat. Discipl.,* 7:223–232, 1969.
19. Steiner, J.E.: The gustofacial response: observation on normal and anencephalic newborn infants. In Bosma, J.F. (Ed.) *Fourth Symposium on Oral Sensation and Perception,* Washington, Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 254–278.
20. Steiner, J.E.: Innate discriminative human facial expressions to taste and smell stimulation (Discussion paper), *Ann. N.Y. Ac. Sci.,* 237:229–233, 1974.
21. Steiner, J.E.: Testing of the senses of taste and smell in craniofacial abnormalities. Paper presented at the Annual Meeting of the American Cleft Palate Association, Boston, Mass., 1974.
22. Steiner, J.E. and Maller, O.: The palatability of food and human chewing habits. *Proc. V. Internat. Sympos. on Physiol. of Food and Fluid Intake.* Jerusalem, 1974, p. 131.
23. Steiner, J.E. and Finnegan, L.: Innate discriminative facial expression to food-related odorants in the neonate infant. *Isr. J. Med. Sci.,* 11:858, 1975.
24. Steiner, J.E. and Finnegan, L.: Innate discriminative facial expressions of the neonate infant to food-related odorants. *Brain, Behavior and Evolution,* (submitted for publication) 1976.
25. Steiner, J.E. and Davies, E.: The functional state of the sense of taste in peripheral circulatory disturbances (in preparation).
26. Wiener, D.: The influence of cigarette-smoking on the human sense of taste. DMD-Thesis Hebrew University Jerusalem, Hadassah School of Dental Med., Jerusalem, Israel, 1968.

DISCUSSION

Moskowitz: There is one problem. In Moncrieff's whole series of rankings for 200 or so odors, he finds that children under six seem to be fairly indifferent.[1]

Steiner: Not only is the study of Moncrieff known to me, but I am also aware that Dr. Engen, who is among the contributors to this volume, is of a similar opinion. In contrast to their views, based on my own observations, as presented here, it is apparent to me that the neonate can differentiate between odors acceptable or pleasant to him and those which are offensive. This differentiative ability is the substrate for elicitation of the reflex-like

[1] Moncrieff, R.W.: *The Chemical Senses* (Rev. ed.) Cleveland, Chemical Rubber Company, 1967.

behavioral signs I described. Furthermore, I am aware of the fact that the neonate infant is exposed, from the very first hours of life, to influences (one might label these even pressures) which can modulate very markedly the infant's preferences. Very soon, perhaps at the age of several weeks or months, the developing infant will display preferences towards chemical stimuli he perceives, resulting from two components a) the innate (non-learned) ones, which are reflected at birth by the facial play and b) the learned ones. The so often described preferences and aversions respectively, of young children towards certain food, is composed of the above mentioned factors.

As to the communicational value of the facial expressions in response to taste and odor stimuli, I would like to refer to another observation. Projecting slides or motion pictures of the neonate's facial expressions to an audience I have noticed that while watching the projected pictures many adults respond spontaneously by mimicking the projected grimaces. This "mirror-grimacing" (resembling the "contageous" properties of a yawn in a classroom) speaks for the communicational value of these basic expressional tools.

PFAFFMANN: In the rat and in animals, the olfactory system is still at the time of birth only partially differentiated. There is a lot of actual migration of the neurons in the olfactory bulb to their appropriate locations. Now, what is the state of the olfactory bulb in the neonate human?

STEINER: As far as I recall, the structure of the olfactory bulb in the human embryo can be clearly seen at the gestational age of six weeks. At this stage the bulb is still hollow and communicates dorsally with the lateral ventricle of the brain. Then the growing stalks become more elongate and the bulb loses its lumen. I think it is hard to point to the particular developmental stage, when the bulb can be seen as a functional unit mature for all its complex tasks, as we know this structure from its functioning in the adult human (or at least in the adult animal from electrophysiological evidences).

PFAFFMANN: Oh well, that may be. But what I'm talking about is the internal structure of the olfactory bulb and I would guess that actual migration and functional development is continuing after birth. That is an important point we ought to be clear about because it suggests that in fact those olfactory pictures are nothing as impressive as your gustatory ones. The interrelations between stimuli and actual growth of neurons going on in the early stages is something I think you ought to look at and clarify from a neurological point of view. This is the one part of the brain that is still in an embryonic state in many animals. In these neonatal animals you actually have growth and proliferation of dendrites.

STEINER: Unfortunately, I don't know of any experimental study establishing reliable correlation between structural and functional growth of the olfactory apparatus in man. Such an investigation would, of course, be basic but it would very difficult to carry out.

Chapter 14

FROM REFLEX TO REPRESENTATION: TASTE-ELICITED TONGUE MOVEMENTS IN THE HUMAN NEWBORN

Geoffrey H. Nowlis

This paper presents some data on the effects of various sugar solutions on tongue movements during human neonatal sucking in the context of three theoretical notions. This context is offered in the spirit of the great mathematical physicist, Sir Arthur Eddington, who cautioned, "Never trust a fact unless it is supported by theory."

The first notion is that those three cranial nerves whose visceral sensory branches have maintained throughout phylogeny a chemosensitive function are, in the vertebrates, the afferent limbs of alimentary reflexes of intake and rejection. Of these three nerves, the tenth, ninth and seventh, the tenth has already been shown to provide the chemosensitive afferents innervating reflex stomach emptying.[2] I have proposed [13] that the chemosensitive branch of the ninth nerve is the afferent limb of an oral rejection reflex by which the contents of the *mouth* are expelled by the tongue. The seventh nerve is the chemosensitive branch of the afferent limb of an intake reflex by which fishes take food into the mouth.[7] I have proposed [13] that in higher vertebrates the chemosensitive branch of the seventh nerve is the afferent limb of an intake reflex in which food is propelled from the front of the mouth back toward the receptive field for swallowing in the pharynx. The palatability of a given gustatory stimulus can then be defined as the relative degree to which these two reflexes of oral intake and rejection are elicited by that stimulus, a balance which is in turn dependent on the different sensitivities of the seventh and ninth nerves to different tastes.

The second notion is that one of the ways that the hypothalamic regulatory centers express their control over food intake is by selectively facilitating or inhibiting one reflex over the other, producing a predisposition toward intake in the case of hunger, and toward rejection in the case of satiation. If this is the case, we should ex-

Acknowledgment: Portions of this paper were presented at a previous symposium.[12] The research was supported by Nutrition Foundation Grant # 440 to William Kessen and J. Jay Braun, with additional funds from NIH Grant USPHS 5 RO1 HD-0890-14, to William Kessen, and NIH Grant USPHS NS 08658, to J. Jay Braun. The author is grateful to: Drs. Alexander Campbell and Joseph Warshaw, responsible physicians for the project; Mr. Gregory Glass, who very ably assisted in the collection of data; and Mr. Hans Graichen and Mr. Roger E. Avery, who assisted immensely with the design and construction of various versions of the recording nipple.

pect the oversensitivity to taste that has been observed in the hyperphagic hypothalamically lesioned rat [5] and in obese humans.[12] That is, without rostral control over such reflexes, the decision to ingest or not to ingest should be determined rather strictly by the stimulus activating the reflexes. In this circumstance, a gustatory stimulus which activates an acceptance reflex to a greater extent than a rejection reflex should be ingested, as long as ingestion remains possible. Similarly, a gustatory stimulus which elicits a rejection reflex to a greater extent than it does an acceptance reflex should determine quite strictly that the substance not be ingested. If such reflexes, in the normal intact organism, are under the tonic control of the hypothalamus, they might be inhibited or facilitated according to information from blood chemistry allowing ingestive behavior to be modulated according to the state of the organism and freeing behavior from strict determination by the properties of the peripheral stimulus.

The third notion is that complex behavior toward palatable and unpalatable substances, behavior involving choice and judgment, might be based on simple, compelled reflexes of acceptance and rejection like the ones I shall be describing. In its broadest context, it amounts to an extremely simple neurological model of affect or emotion. For the moment, we may view it as an hypothesis concerning the way in which adaptive reflexes of the brainstem, such as those involved in feeding, drinking, fighting, fleeing, and sexual behavior, may become represented as uniquely defined states of the nervous system in the activity of neurons which inhibit the specific reflexes which are elicited by the current stimulus situation.

The reflex is a much maligned concept among those who wish to understand the behavior of human beings and other advanced vertebrates. Very little of the adult intact mammal's behavior seems to be as automatically compelled by an environmental stimulus as would seem implicit in the notion of a reflex. Yet it is interesting that many of the affective judgments that we make, such as that one food tastes good and another tastes bad, seem to be as certainly and unavoidably compelled as are the more overt behavioral manifestations of a reflex. I shall contend that all that is logically necessary to transform a compelled reflex, such as a reflexive rejection movement of the tongue, into an affective judgement that its eliciting stimulus tastes bad, is to inhibit it. This will follow from the observation that in order to turn off one part of the nervous system, another part of the nervous system has to turn on. Thus, the neural activity required to inhibit reflex rejection would represent an affectively negative judgement of the stimulus while that which inhibits an acceptance reflex would represent an affectively positive judgement. The development of the capacity to make such transformations would seem to be one of the most important steps in the behavioral development of the mammalian nervous system. One of the main purposes of this paper is to suggest that the ideal behavioral system in which to study this aspect of neurobehavioral development is that system whereby we categorize gustatory stimuli as to-be-ingested or to-be-rejected.

SOME VERTEBRATE FEEDING MECHANISMS

Shortly after I arrived at the notion of two mammalian tongue reflexes of acceptance and rejection, I came across the remarkable behavioral and neuroanatomical work on ingestive reflexes in fishes [7] begun in the late 1800's by the great comparative neurologist, C. Judson Herrick. I have yet to encounter as thorough and insightful an analysis of the anatomical basis of behavior. Beginning with the observation of taste buds distributed on the outer surface of the body in many species of fishes, Herrick turned to an investigation both of the central connections of these taste buds and of the behavioral significance of the particular location of the taste buds. All such cutaneous taste buds were shown to connect with the brainstem via the seventh cranial nerve, whether they were located in the lips, the fins, the whisker-like barblets of the catfish, or the pectoral fins of the cod, and related species. This is the nerve which innervates the taste buds of the anterior two-thirds of the mammalian tongue. The taste buds on the fish's tongue, which is roughly homologous to the posterior one-third of the mammalian tongue, are connected to the brainstem via the ninth cranial nerve. The fibers of these two nerves enter at different levels a distinct fiber bundle known as the solitary tract.

In a series of behavioral studies, Herrick demonstrated that for each configuration of external taste buds, there existed a different reflex of food intake. For example, in the catfishes, stimulation of the barblets with either a tactile or a gustatory stimulus elicited a lateral movement of the body in the direction of the stimulus and an opening and closing of the mouth on the stimulus source. Careful experimentation demonstrated that this reflex was independently elicited by tactile and gustatory stimulation. In the codfish, when the taste buds of the pectoral fins were stimulated, there was a reversal of forward movement and a downward movement toward the source of stimulation. For these, and for several other configurations of intake reflexes in other fishes, Herrick demonstrated connections between the seventh nerve taste fibers and the appropriate motor neurons for the specific intake movements. The important point for this discussion is that, no matter what the configuration of the reflex, all reflexes of intake had as their afferent limb the seventh cranial nerve. The primitive tongue of fishes has taste buds innervated by the ninth cranial nerve and is apparently used primarily for rejection. In species adapted for bottom feeding, mouthfuls of mud and debris are taken in along with potential food, most of which must be rejected. These species have evolved an enormous palatal organ, with taste buds innervated by the ninth nerve, to accomplish this rejection.

Taste buds on the tongue of reptiles and amphibians are usually innervated by the ninth nerve. In mammals, a new adaptation for feeding has been added in the form of the anterior two-thirds of the tongue. This is an exquisitely mobile organ which serves to position food [6] with respect to the teeth and with respect to the receptive field for swallowing,

further back in the pharynx.[10,21] With no external taste buds, the behavioral decision to accept or reject food on the basis of taste must, in the mammal, employ the tongue. It is perhaps no accident that this new portion of the tongue has its taste buds innervated by the seventh nerve, with its phylogenetic history of sensitivity to substances appropriate for ingestion.

The relevant human neuroanatomy is schematically represented in Figure 14–1. Gustatory information from the seventh nerve enters the rostral por-

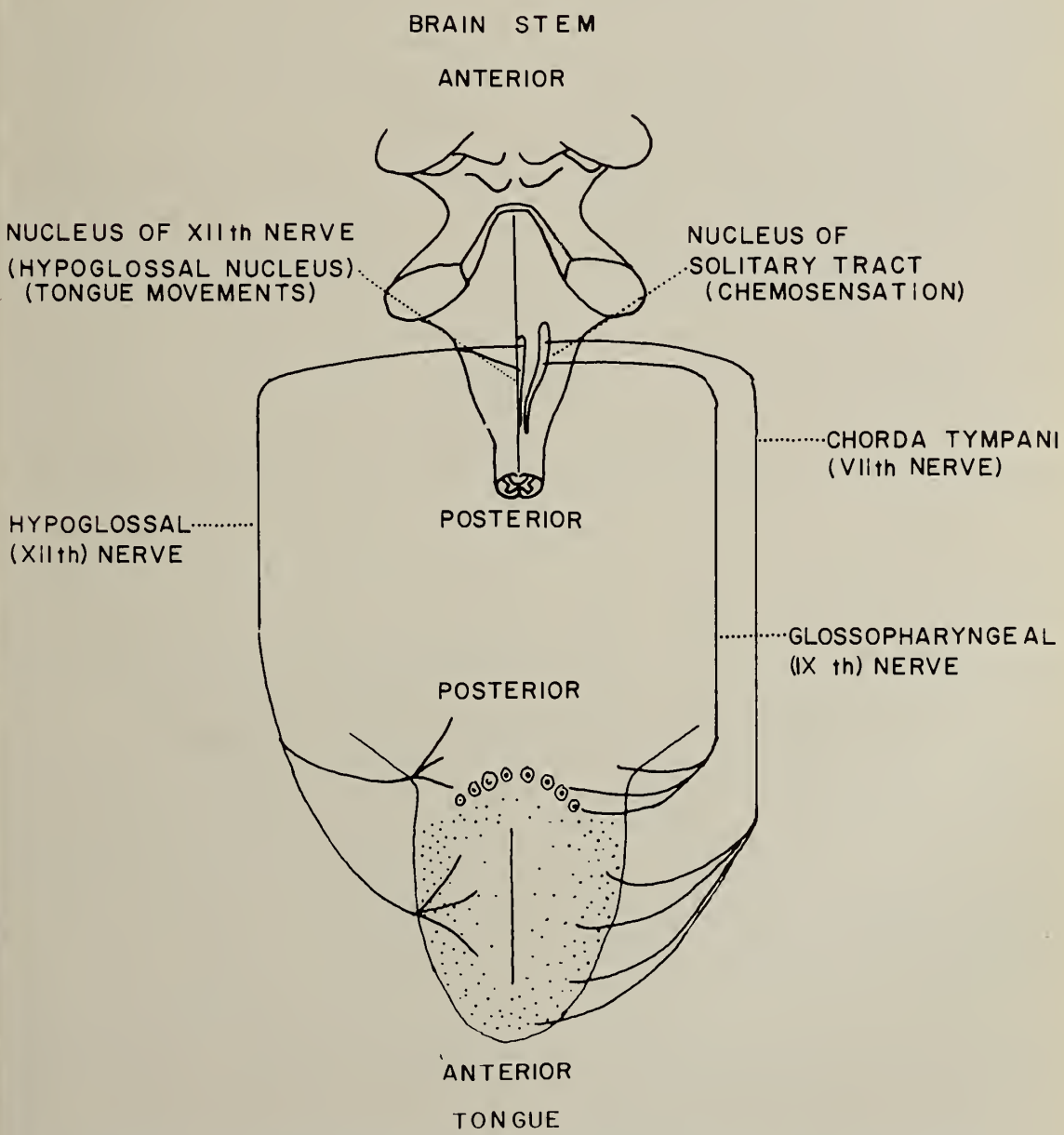


Figure 14–1. Schematization of structures which may be involved in taste-elicited tongue reflexes.

tion of the nucleus of the solitary tract. Further back, the gustatory information from the ninth nerve is represented. Directly ventral and medial to this sensory nucleus lies the hypoglossal nucleus, responsible for motor innervation of the tongue. Electrical stimulation of the rostral portion of this motor nucleus in the cat evokes an inward movement of the tongue, while stimulation further back elicits tongue protrusion.[8]

Although both of these gustatory nerves along with the tongue areas they supply are responsive to some degree to each of the four basic tastes, there appear to be systematic differences in their relative sensitivity. Thus, according to classical accounts,[22] the tip of the tongue is most sensitive to sweet, and further back, but still within the receptive zone of the seventh nerve, it is most sensitive to salt. Still further back, sour is more strongly represented. Once the transition is made to the area of the ninth nerve, bitterness predominates. Thus, both in terms of the anterior-posterior distribution within the seventh nerve receptive field, and in terms of the more distinct transition from the seventh to ninth nerve zones, the input to the nucleus of the solitary tract, following the topographic organization of the tongue, is biased rostrally in favor of sweet and salt, which are preferred in moderately concentrated aqueous solutions to plain water by most species, and caudally in favor of bitter and sour, to which water is generally preferred.[18] The reflex theory of palatability suggests that to the extent that gustatory impulses are sent to the rostral portions of the nucleus of the solitary tract, intake activity should be evoked. To the extent that gustatory impulses are sent to the caudal portions of this nucleus, rejection activity should be evoked. Thus, the activity evoked by a given gustatory stimulus in the seventh nerve, weighted according to the anterior-posterior location within this receptive zone of the maximal sensitivity for this taste, minus the activity evoked by this stimulus in the ninth nerve, should yield a quantitative estimate of the palatability of the stimulus. I shall illustrate this procedure later.

Philip Bard and his student Martin Macht have studied the feeding behavior of the chronic decerebrate cat,[1,9] a preparation left with basically the remnant of the brain illustrated in Figure 14–1. Immediately after decerebration, food placed on the front of the tongue elicits no behavior. After about two weeks, however, palatable food such as a chunk of liver is taken further into the mouth by a movement of the tongue and elicits reflex swallowing. Adulteration of these liver chunks with various taste solutions causes the liver to be expelled from the mouth instead of being swallowed, reportedly at the same concentrations at which an intact cat avoids these tastes.

I have electrically stimulated the seventh and ninth nerve areas of the tongue of decerebrate cats and regularly elicited movements of the tongue which conform to my notion of an acceptance and a rejection reflex. Stimulation of the ninth-nerve-innervated circumvallate papillae elicited an outward movement of the tongue with an upward arching of its posterior segment. Stimulation of the anterior portions of the tongue elicited a movement in

which the tip and edge of the tongue curled upward ipsilaterally and an inward movement of the whole tongue. The stimulation in these instances was as likely of somatosensory as of gustatory receptors, but this work will be continued with cats which have survived the two weeks necessary for the return of gustatory responsiveness. Preliminary studies in decerebrate rats indicate that taste responsivity may be recovered earlier in this species than in the cat.*

TASTE RESPONSE IN THE HUMAN NEWBORN

Against the background of these observations, William Kessen and I began a study of taste-elicited movements of the anterior and posterior tongue in one- and two-day old human babies.[16] Since so many reflexes are present in newborns which disappear during maturation, such a study seemed an ideal way to test the relevance of the reflex model of palatability to human beings. We also felt that it would provide a potential psychophysics of these two areas of the newborn's tongue, and that a comparison of the psychophysical functions for anterior and posterior tongue could yield an estimate of the infant's preferences and aversions for various tastes.

Our need to record pressures independently from the front and back of the tongue imposed several constraints on the form of the pressure-transducing nipple we have been developing. First, in order to ensure independent compression of the two sensitive areas of the nipple, it had to be relatively rigid. Second, in order to measure the pressure from the back of the tongue, the nipple had to be long enough to protrude rather deeply into the infant's mouth. Figure 14–2 shows a highly schematized drawing of the head and oral cavity of a newborn infant, derived from sketches and castings of the oral cavity of a cadaver. The arrow indicates the approximate point of transition between the anterior and posterior portions of the tongue of the newborn, beyond which the nipple must intrude. Third, in order not to elicit gagging and the accompanying rejection movements of the tongue with the tactile stimulation from the rigid nipple itself, it had to be made as unobtrusive as possible, thus accounting

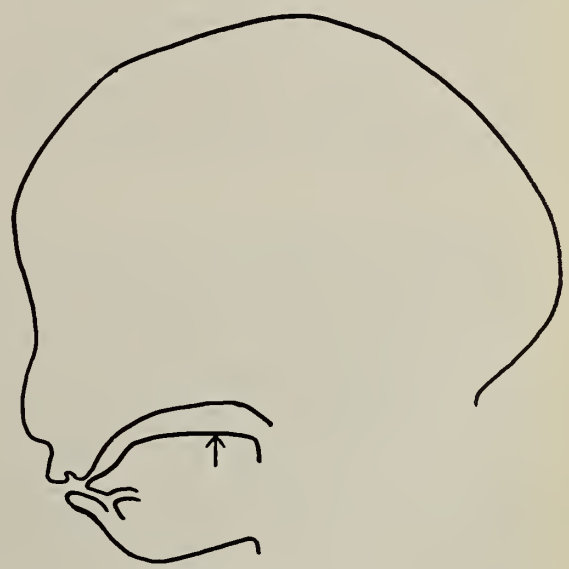


Figure 14–2. Schematized mid-saggital view of newborn infant's head, showing tongue in oral cavity. Recording nipple apposes palate and protrudes into mouth as far as arrow.

*Among the few decerebrate rats tested to date, small amounts of applesauce placed in the mouth 2–3 days post-operatively elicited tongue and jaw movements whereby some of the applesauce was eventually swallowed. Applesauce adulterated with quinine appeared more likely to end up outside the mouth. These observations require repetition and quantification.

for the tapering tip and the dorsal arch of the nipple, designed to conform to the shape of the palate (see Figure 14–3). In addition to the three tubes shown in Figure 14–3, the nipple has a fourth tube for delivery of fluids, parallel to the one for measuring negative pressure in the mouth. The system is arranged so that negative pressure in the infant's mouth delivers the fluid to the mouth.

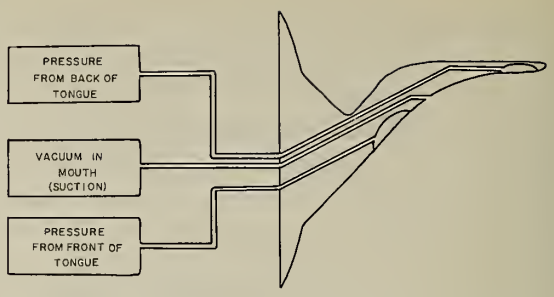


Figure 14–3. Diagram of recording nipple.

Figure 14–4 is a sample of data from the three recording channels of the nipple, obtained while the infant was offered the standard hospital formula. The main point of this figure is to demonstrate that the two tongue pressure channels are measuring events whose peak amplitudes are offset from one another in time. This is an important rationale for speaking of two different tongue responses. A burst of sucking may begin with a few low amplitude sucks typically followed by stronger and stronger sucks, building up to a peak amplitude which is maintained for a number of sucks. The amplitude then decreases for the final few sucks of the burst.

We have found consistently that more concentrated sugar solutions elicit anterior tongue movements of greater peak amplitude than do less concentrated solutions, and that different sugars vary in their capacity to affect tongue pressure. This situation is summarized in Figure 14–5. The four stimuli employed were 2% (0.058M) and 4% (0.117M) sucrose,

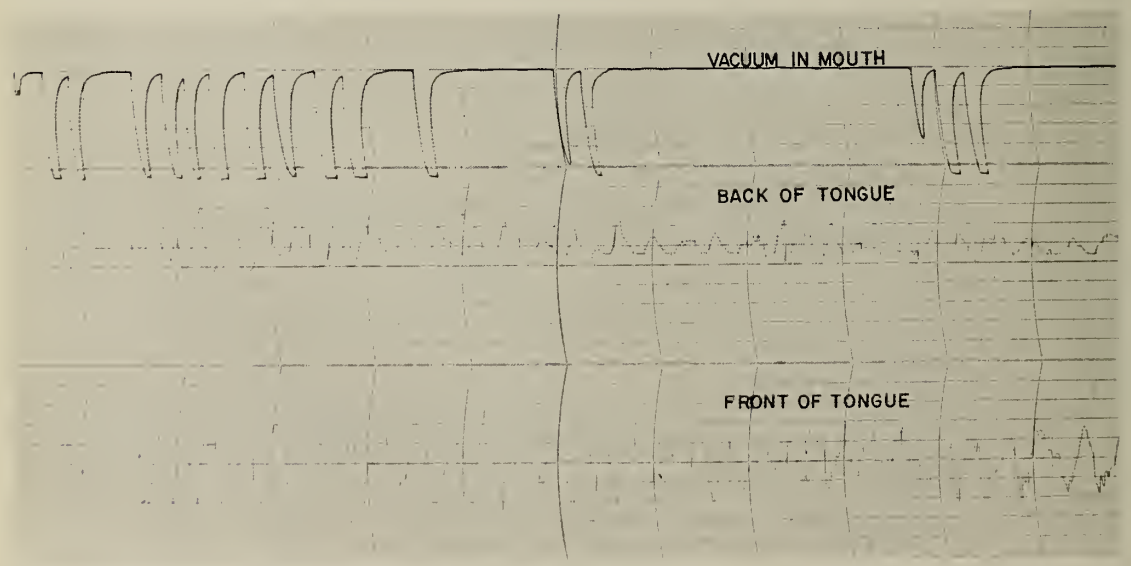


Figure 14–4. Sample of pressure variations from front and back of tongue and vacuum in mouth during newborn's sucking while being fed standard hospital formula. Two horizontal divisions equal one second. Vertical axis is not calibrated.

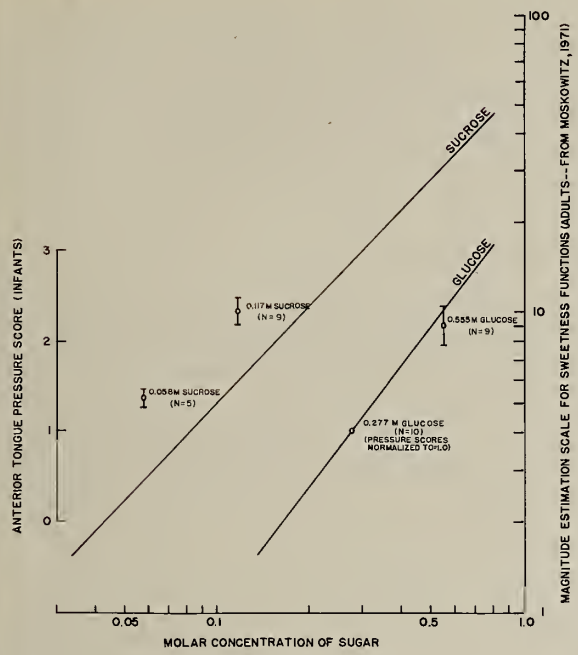

ANTERIOR TONGUE PRESSURE SCORE (INFANTS)
MAGNITUDE ESTIMATION SCALE FOR SWEETNESS FUNCTIONS (ADULTS--FROM MOSKOWITZ, 1971)
SUCROSE
GLUCOSE
0.058M SUCROSE (N=5)
0.117M SUCROSE (N=9)
0.277 M GLUCOSE (N=10) (PRESSURE SCORES NORMALIZED TO 1.0)
0.555M GLUCOSE (N=9)
MOLAR CONCENTRATION OF SUGAR

Figure 14–5. The four data points are tongue pressure scores (left ordinate) for the four sugar solutions given to infants. The two lines (from Moskowitz [11]) are adult psychophysical functions (right ordinate). From Nowlis and Kessen.[16]

and 5% (0.277M) and 10% (0.555M) glucose. Using 5% glucose as a standard, we tested seventy-five healthy full-term infants with a variety of gustatory stimuli. The data summarized in Figure 14–5 are from all infants who received at least one other sugar concentration in addition to the standard. The anterior tongue pressure score is derived by taking the mean amplitude of the strongest ten sucks to a given concentration, and dividing that by the mean amplitude of the strongest ten sucks to the standard glucose concentration for that infant.

Two features of these data are immediately apparent. First, amplitude of anterior tongue pressure is for both sugars a direct function of sugar concentration; as sugar concentration doubles, there is a near doubling of tongue pressure. Second, at least at the concentrations we employed, sucrose is a more effective stimulus than glucose. A comparison with adult responses to varying concentrations of these two sugars can be derived from the two sweetness magnitude estimation functions[11] superimposed on Figure 14–5. There is a remarkable similarity in slope and relative height between the psychophysical curves and those implicit in the four data points from the infant tongue pressure responses. One of the advantages of this pressure measurement technique is that we can get good information from our subjects with a very brief sample of behavior, and hence can limit the amount of a given stimulus offered for tasting to two milliliters. This means that we can test a single infant with a number of different stimuli in a relatively brief session.

When a 10^{-4} M solution of quinine hydrochloride is offered, the bout of rhythmic sucking is disrupted within a few sucks. This is usually followed by isolated movements of the posterior tongue which exert a sustained pressure against the nipple for one or two seconds. We have not yet explored many different concentrations of unpleasant tasting stimuli. One reason is that the current version of the recording nipple is most sensitive to those movements of the posterior tongue which are directed upward, toward the palate, whereas the gross movements of rejection we can feel as we hold the nipple in the infant's mouth are definitely and consistently directed outward, tending to push the nipple out of the mouth. This particular movement registers only feebly

and inconsistently with the current version of the nipple, which is being modified to record the rejection movements more accurately. However, by producing mixtures of quinine and sucrose, we have been able to elicit a rhythmic sucking in some infants in which the posterior tongue participates well and the anterior tongue participates little, if at all, according to our pressure measurements.

FROM REFLEX TO PALATABILITY

The manner in which we hope to combine the measurements from the front and back of the infant's tongue to yield an estimate of palatability can be illustrated with physiological and behavioral data from the work of Pfaffmann and his colleagues with the laboratory rat. Preference-aversion curves are derived using the two-bottle preference test,[20] in which one of the available bottles contains plain water. The rat is said to prefer the solution to plain water if it drinks more than fifty percent of its daily fluid from the bottle containing that substance, or an aversion for that substance if less than half of its total fluid intake is from that bottle. Figure 14–6 [18] depicts such preference-aversion curves for four basic gustatory stimuli. Figure 14–7 compares the integrated whole nerve activity from the rat's seventh nerve (chorda tympani) and ninth nerve (glossopharyngeal) nerve, from studies by Pfaffmann, Fisher and Frank.[19] Let us concentrate for a moment on the curves for sodium chloride.

Notice that in response to sodium chloride the activity in the ninth nerve begins to increase at a higher concentration than does that in the seventh nerve, but the ninth nerve activity increases at a steeper rate. Thus, at lower concentrations of salt, there is more seventh nerve activity, while at higher concentrations, there is more ninth nerve activity. Figure 14–8 illustrates the simple procedure of subtracting the curve of ninth nerve activity to salt from that of the seventh nerve activity. When the difference between the two curves is positive

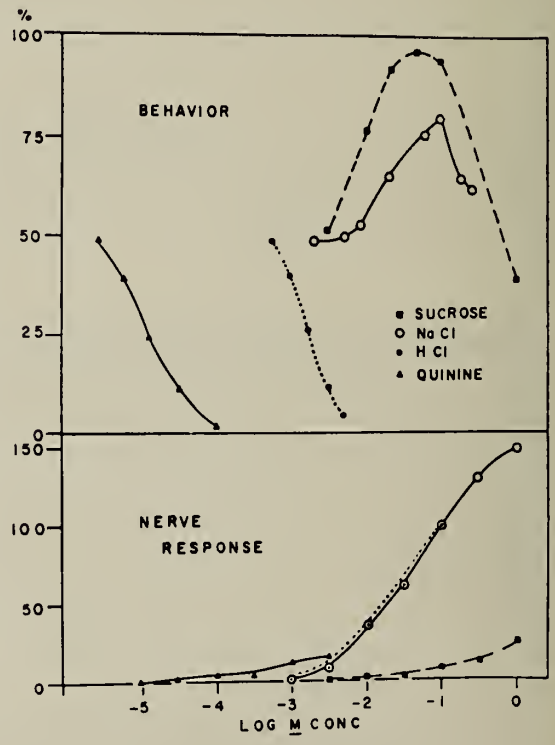


Figure 14–6. Composite graph of preference-aversion functions and integrated whole chorda tympani responses in the rat. The upper half shows percentage intake of the taste solution as a function of concentration. The lower half shows chorda tympani responses in arbitrary units. (From Pfaffmann [18])

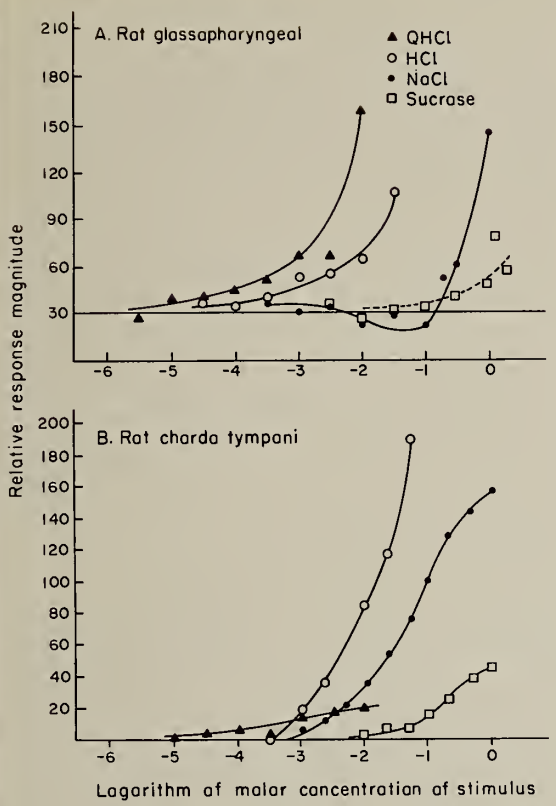


Figure 14–7. Composite graph showing integrated whole nerve responses in arbitrary units from rat glossopharyngeal nerve (top half) and rat chorda tympani (lower half), as a function of concentration of solutions of quinine hydrochloride (bitter), hydrochloric acid (sour), sodium chloride (salty), and sucrose (sweet). (From Pfaffmann, Fisher, and Frank[19])

(seventh greater than ninth) this difference is plotted above the fifty percent, or neutral palatability level, as an increment in palatability. When the difference is negative, it is plotted below the fifty percent level, as a decrement in palatability. The resultant theoretical curve bears a remarkable resemblance to the sodium chloride preference-aversion curve for the rat shown in Figure 14–7. This simple method can produce theoretical curves which correspond quite well to the empirical palatability curves if one assumes that the important variable is the rostral-caudal focus within the solitary tract, as discussed earlier, so that the contribution of the seventh nerve for a particular taste should be weighted more positively the further anteriorly that taste is represented on the tongue (and hence, presumably, in the solitary nucleus).

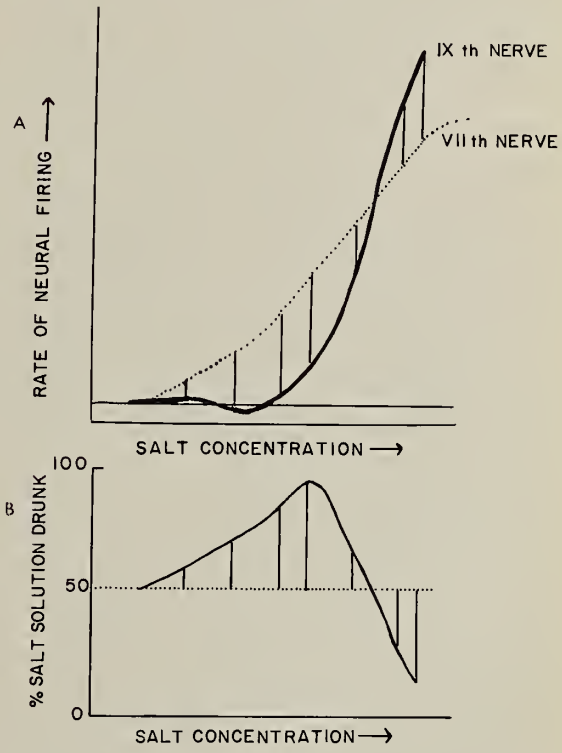


Figure 14–8. Schematization of derivation of rat preference-aversion function for sodium chloride from neural activity functions.

A. Integrated whole nerve responses in seventh and ninth nerves as a function of concentration of sodium chloride, replotted from data presented in Figure 4 of Pfaffmann et al.[19]

B. "Preference-aversion" function for sodium chloride, derived by subtracting ninth nerve activity from seventh nerve activity at the points indicated by vertical lines.

If these palatability functions are, indeed, based on a comparison of information from the seventh and ninth nerves cutting one nerve or the other should eliminate the positive or negative portion of these functions. That this is not the case in otherwise intact rats was demonstrated some years ago.[17] Preference functions were not appreciably altered unless both seventh and ninth nerves were cut. One likely reason for this is that intact mammals very quickly learn alterations in their preferences and aversions for tastes. Many of these learned palatability alterations have been recently shown to be eliminated (and many relearnable), following removal of the gustatory cortex.[3] It is reasonable to assume that, as the organism experiences reflexive rejections and acceptances of various tastes, it learns the reflexive hedonic value of given concentrations of given tastes. Using the technique of Garcia, I have shown [14] that a single association of a given concentration of salt with a nauseating event produces a learned aversion which, although it generalizes to other concentrations, has nonetheless a significant specificity *to that concentration* of salt. Information about the concentration of tastes reaches the cortex via either the seventh or ninth nerve. If the reflexively based preferences and aversions are associated with the representation of their concentrations in the gustatory cortex, we might expect the animal with intact cortex to express this now learned preference with information about concentration from only one of the nerves.

It was with this notion in mind that I began, with J. Braun, a series of studies on the palatability behavior of rats with gustatory cortex removed. Although we have not yet done the study involving cutting the seventh or ninth nerve, I would like to mention some of the results of our first study,[15] because they indicate quite strongly that the basic nerual circuitry for preferences and aversions exhibited by the intact rat is sub-cortical. We compared the preference-aversion functions of two groups of rats, one intact and one with the gustatory cortex bilaterally removed. We used a fifteen-minute one-bottle preference test in this study because we reasoned that if the memory for which bottle contained which solution in a two-bottle test were at all dependent on an intact cortex, we would be stacking the cards against our animals with cortical ablations being able to express their preferences if we used two-bottle tests. The results were quite clear. Although some systematic differences appeared, the animals with cortical ablations showed very normal looking preference and aversion curves, ones which resembled not only the curves of the intact rats, but also were strikingly similar to both the theoretical curves and the empirical functions for intact rats based on the two-bottle choice situation illustrated in Figure 14–6.

Interestingly the older parts of the rat's brain are capable of producing preferences and aversions which are essentially similar to those from rats with an intact brain. It is equally interesting that one- and two-bottle tests produce such similar results. The only means by which a rat can express its preferences in a one-bottle test is by either changing its rate of lapping, or by stopping sooner or later in the brief test period. In the two-bottle

test, amount drunk of the flavored solution is dependent not only on these variables, but also on the decision to return to a given bottle which some minutes or hours ago was discovered to contain a palatable substance, or to search out that bottle when water is discovered in the other. The similarities in the preference and aversion functions based on both these techniques, and between these and those from rats lacking gustatory neocortex, suggests to me that there is some common neural event underlying the preference behavior in all of these empirical situations. I would suggest that that neural event is a monitoring of the extent to which tongue reflexes of acceptance and rejection are elicited by the various gustatory concentrations involved. Similarly, I suggest that the crucial neural event in producing the basis for human adult palatability judgements is a monitoring of the magnitude of an anterior tongue reflex demonstrable in newborn humans. But how can this be so, since these reflexes are obviously not an overt part of adult taste and palatability judgements?

A MODEL OF AFFECTIVE REPRESENTATION

Many reflexes are present in the newborn which disappear with maturation, yet reappear as signs of neurological damage. One strong inference is that the disappearence of these reflexes is simply a function of inhibitory control from fore-brain functions. The exciting observation we can make is that in order to inhibit such a reflex, it is necessary that there be a set of neurons whose activity is responsible for the inhibition of the reflex. The more strongly the reflex is elicited, the stronger must be the activity of these specific inhibitory neurons in order to inhibit the compelled behavior. The activity of these specific inhibitory neurons, then, is necessarily an analogue representation of that reflex that would be elicited, were it not being inhibited, be it a feeding, fighting, or sexual reflex. In freeing the organism from the compelled behavior, the inhibitory mechanism transforms the compelled reflex into information as to which types of behavior are relevant. In short, the activity of the inhibitory neurons preserves a behavioral distinction in a nonbehavioral form. To the extent that I am inhibiting a tongue rejection reflex, there exists an activity in my nervous system that corresponds to the information that something bad tasting is in my mouth. To the extent that I am inhibiting an intake reflex, there exists an activity in my nervous system that corresponds to the information that something good-tasting is in my mouth. Yet now, instead of curling up my tongue to propel it down my gullet, I am free to savor it, share it, or assign a number to it. Certainly the development of such a representational capacity is one of the most important processes in the developing human being.

The reason that I would argue that the palatability reflex system is the ideal one in which to study the phylogenetic and ontogenetic development of such a representational capacity is that measurements of the dependence of these reflexes on taste concentration produces quantitative functions of such distinctive form that

they can easily be recognized when manifested in behavior, like choice and judgment, which is dependent on a neural representation of the behavioral tendencies. As the great comparative anatomist, J.Z. Young [23] observed, "No animal can live without food. Let us then pursue the corollary of this: namely that food is about the most important influence in determining the organization of the brain and the behavior that the brain organization dictates."

REFERENCES

1. Bard, P., and Macht, M.B.: The behaviour of chronically decerbrate cats. In Wolstenholme, G.E.W., (Ed.) *Ciba Foundation Symposium on the Neurological Basis of Behavior.* Boston, Little Brown, 1958, 55–75.
2. Borison, H.L., and Wang, S.C.: Physiology and pharmacology of vomiting. *Pharm. Rev.* 5: 193–230, 1953.
3. Braun, J.J., and Ouellet, J.V.: Personal communication, 1974.
4. Brizzee, K.R.: Effect of localized brain–stem lesions and supradiaphragmatic vagotomy on irradiation emesis in the monkey. *Amer. J. Physiol.* 187: 567–570, 1956.
5. Corbit, J.D., and Stellar, E.: Palatability, food intake, and obesity in normal and hyperphagic rats. *J. comp. physiol. Psychol.* 58:63–67, 1964.
6. Davis, D.D.: Origin of the mammalian feeding mechanism. *Am. Zool.* 1: 229–234, 1961.
7. Herrick, C.J.: The organ and sense of taste in fishes. *Bull. U.S. Fisheries Comm. for 1902,* 1903.
8. Kawamura, Y., and Morimoto, T.: Neurophysiological mechanisms related to reflex control of the tongue movements. In Bosma, J.F., (Ed.) *Fourth Symposium on Oral Sensation and Perception.* Washington, Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 206–217.
9. Macht, M.B.: Subcortical localization of certain "taste" responses in the cat. *Fed. Proc.,* 1951, 10: 88 (abstract).
10. Miller, F.R., and Sherrington, C.S.: Some observations on the buccopharyngeal stage of reflex deglutition in the cat. *Quart. J. Exp. Physiol.* 9: 147–186, 1916.
11. Moskowitz, H.R.: The sweetness and pleasantness of sugars. *Am. J. Psychol.* 84: 387–405, 1971.
12. Nisbett, R.E.: Taste, deprivation, and weight determinants of eating behavior. *J. pers. soc. Psychol.* 10: 107–116, 1968.
13. Nowlis, G.H.: Taste-elicited tongue movements in human newborn infants: An approach to palatability. In Bosma, J.F. (Ed.) *Fourth Symposium on Oral Sensation and Perception,* Washington, Superintendent of Documents, U.S. Government Printing Office, 1973. pp. 292–303.
14. Nowlis, G.H.: Conditioned stimulus intensity in acquired alimentary aversions in the rat. *J. comp. physiol. Psychol.* 86: 1173–1184, 1974.
15. Nowlis, G.H., and Braun, J.J.: Preferences and aversions for gustatory stimuli in rats after gustatory neocortical ablation. (In preparation).
16. Nowlis, G.H., and Kessen, W.: Human newborns differentiate differing concentrations of sucrose and glucose. *Science,* 191:865–866, 1976.
17. Pfaffmann, C.: Taste preference and aversion following lingual denervation. *J. comp. physiol. Psychol.* 45: 393–400, 1952.
18. Pfaffmann, C.: The pleasures of sensation. *Psychol. Rev.* 67: 253–268, 1960.
19. Pfaffmann, C., Fisher, G.L., and Frank, M.E.: The sensory and behavioral factors in taste preferences. In Hayashi, T., (Ed.), *Olfaction and Taste.* New York: Pergamon, 1967, pp. 361–371.
20. Richter, C.P.: Total self-regulatory functions in animals and human beings. *Harvey Lectures,* 38: 63–103, 1942.

21. Storey, A.T.: Laryngeal initiation of swallowing. *Exp. Neurol.* 20: 359–365, 1968.
22. Woodworth, R.S. and Schlosberg, H.: *Experimental Psychology.* New York. Holt, 1954.
23. Young, J.Z.: Influence of the mouth on the evolution of the brain. In Person, P., (Ed.) *Biology of the Mouth.* Washington, AAAS publication No. 89, 1968, 21–35.

DISCUSSION

Le Magnen: What is the part, in your opinion, of olfactory stimuli in these reflexes?

Nowlis: I can respond to that question only at the conceptual level, although I hope soon to be able to approach it neuroanatomically. For the moment, I will simply guess that those flavors (with an olfactory component) which enhance intake activate the olfactory bulb as well as the taste buds, and that the influence of this olfactory input is either a simple facilitation of the intake reflex or an inhibition of the rejection reflex. In the case of flavors which decrease palatability, I would expect the reverse: that the olfactory component of the complex stimulus either facilitates the rejection reflex or inhibits the intake reflex. It even seems plausible that one reason so many culinary preparations include a little bit of salt or sugar or other sweetner is to provide a baseline of reflex intake activity which is then enhanced by the olfactory component of the food.

Steiner: I have a criticism that applies to the previous paper, that's mine, as well as to Dr. Nowlis' paper. The criticism is that the indication of acceptance and rejection finally must be done in a quantative way using the consumption or the consummatory behavior. I know that all delicate fine indicators which have been collected point to preference behavior, they are of highest value and interest, of course, but the most important reflection will be if somebody will quantify the consummatory behavior.

Only by this measure or by combining behavioral signs of preference with consummatory behavior in quantitative terms can one finally judge that the organism prefers one substance and rejects the other.

Nowlis: It is precisely with the intention of quantifying that consummatory behavior that I have undertaken a study of these reflexes, since I feel that it is by means of these reflexes that the final decision to ingest or not to ingest on the basis of taste is made.

Steiner: Exactly, therefore, I agree very much. But your recording of sucking and of pressure from different areas of the tongue must later be combined with data pointing to the amounts of food actually ingested. Dr. Owen Maller and myself tried to carry out such a study of ingestive behavior and mastication of different sweetened food in the adult and I have the feeling that we have some common points from the behavioral indicators to real ingestion. This may be a continuum but it must be much more clearly demonstrated.

Nowlis: I think I differ with your relative emphases. When studying any kind of behavior, one usually has a choice of many indicators of that behavior to pursue. To my mind, there is no reason to view the final outcome of a behavior as the optimal entity to quantify. In studying aggres-

sion, for example, I would be at least as happy to see quantification of overt or covert aggressive behavior as I would to quantify the casualties.

If a diet is diluted with a non-caloric filler, most animals will eat more of it in 24 hours than they will of the undiluted diet. Given a choice between the two, however, they may often choose the undiluted diet, unless there are very attractive gustatory or olfactory properties to the dilution material. Which measure should we use for a preference measure? If a man drinks more water in a day than he does tea or Scotch whiskey, shall we conclude that he prefers water over the other two? The act, the behavior of acceptance and rejection can be modified by a number of factors, including so-called post-ingestional factors. Measures of volume ingested are very sensitive to these factors. This is one reason why I seek to devise a technique which needs but a few seconds and a few milliliters per stimulus. Cross-validation between this and other measures of taste sensitivity and preference is essential, but volume ingested may not turn out to be the primary criterial indicator.

Chapter 15

SENSORY MECHANISMS OF THE NEWBORN'S TONGUE

James M. Weiffenbach

The sense of taste is intimately involved in the control of ingestion, but tasting is different from ingesting and the control of ingestion involves factors in addition to taste. To study taste as a sensory modality or to analyze its role in the modulation of intake requires that taste effects and non-taste effects be separated from each other. In studies of animals, taste factors can be isolated by making a taste stimulus the cue, but not the reinforcer, for discriminative behavior.[9] Studies of human adults employ a variety of techniques to maximize taste effects. Stimulation may be limited to specified areas on the dorsal surface of the tongue [2,4,8] or if other mouth receptors are to be involved, arrangements are made to insure that the test fluids are not swallowed. Rather than inferring the adult's taste capabilities from observations of his ingestive responses in a feeding situation, we require that the subject make a non-ingestive indicator response, usually a verbal one. The human newborn is not verbally competent and will not inhibit swallowing on command. However, the infant exhibits an oral reflex that can be cued by a minute drop of fluid applied to a restricted site on the tongue. This potential indicator response is minimally associated with ingestion and can be elicited outside the normal sequence of feeding behavior.

MEASURING SENSITIVITY

The lateral tongue movement

Touching the newborn's tongue on either the right or the left side triggers a distinctive lateral movement of its tip toward that side. The frequency of occurrence of this readily identified, reliably observed, response reflects the intensity of a touch stimulus and is a useful indicator of touch sensitivity in the early days and weeks of life.[12,13,14] A similar ipsilateral tongue motion is elicited by a single drop of fluid placed on the dorsal surface of the tongue near its apex (see Figure 15–1). Since neither the form nor the frequency of occurrence of drop elicited responses varies with the taste

Acknowledgement: I am indebted to Captain David W. Bailey, MC, USN, Chief of Pediatrics, National Naval Medical Center, Bethesda, Maryland, and his staff for their cooperation that has made these investigations possible. I thank Dr. Bradley T. Thach for his contribution to the early studies and thank Miss Sandra Middleton, Mrs. Deborah Barzola and Miss Becki Kerkering for their assistance in the execution of subsequent ones. I am grateful to Dr. Linda M. Bartoshuk for commenting on an early version of the chapter manuscript. The illustrations that appear as Figure 15–1 are the work of Ms. Beverly Etter. I wish to express my appreciation for her contribution.

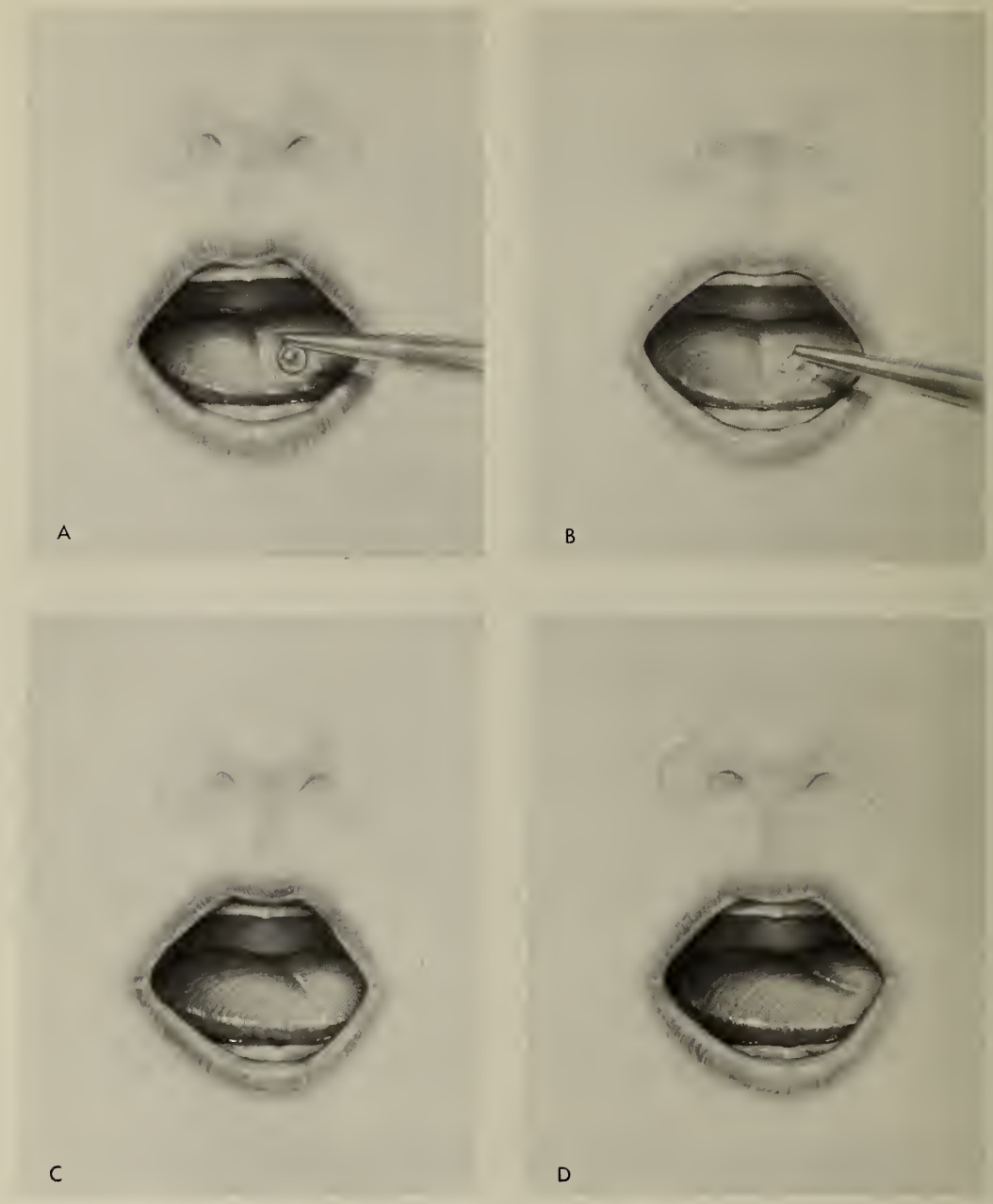


Figure 15–1. Fluid stimuli are applied to the dorsum of the human neonate's tongue at sites to the right or left of the midline near the tip. The infant is on his back with his arms swaddled at his sides. The infant's head is held in a median orientation and the mouth opened by gentle pressure on the chin. A five microliter stimulus drop, adhering to the tip of a micropipette is lowered over a selected site (A). On contact, the drop breaks away from the micropipette to form a localized pool on the moist surface of the tongue (B). The tongue moves from its initial position at midline toward the side of stimulation (C). The response can occur prior to any spreading of the fluid stimulus on the tongue's surface and may continue until the tongue tip is carried far to the ipsilateral side (D). The tongue returns to its initial position at midline without further stimulation.

characteristics of the eliciting fluid, these parameters of response cannot serve as direct indices of taste. However, the response undergoes adaptation, i.e. repeated stimulation leads to reduced frequency of responding. If such adaptation can be shown to be specific to the taste of the stimulus, it will provide both a measure of the taste sensitivity of the newborn's tongue and a basis for inferring the taste receptor mechanisms required to support such sensitivity.[15]

The paradigm

In what has become a standard procedure, normal newborns between one and three days of age, are tested in an only slightly protected nook in one corner of the newborn nursery of a suburban hospital. Infants are swaddled with their arms at their sides and placed on their backs with their heads at midline. At thirty second intervals gentle pressure is applied to the infant's chin. When the infant's mouth is stably open and the tongue centered at rest, a five microliter drop of fluid is transferred from a micropipette to the left side of the infant's tongue (see Figure 15–1). One investigator applies the stimulus while another maintains the infant's head in a midline position. They independently report whether or not they observed a displacement of the tip of the infant's tongue toward the stimulated side. Stimulation with water drops continues until six successive failures to respond cue a shift in stimulus to a test fluid.* Twelve stimulations with the test fluid complete the first trial. The entire procedure is repeated after an interval of no less than 10 minutes. The frequency of response occurrence for the twelve test stimulations of each trial is then compared for infants receiving a tastant test fluid and those receiving water drops throughout.

A demonstration using glucose

This testing paradigm was first used to investigate the sensitivity of the newborn's tongue to an example sweet tastant, glucose.[15] Each of 48 infants received initial stimulation with water drops followed by 12 stimulations with one of four test fluids. Test fluids, water or glucose solution at 0.28, 0.55 or 2.77 molar concentration, were assigned randomly within groups of infants equated for sex and age (6 to 30 and 30 to 72 hours). Each infant received the same test fluid for each of two trials. Comparison of trial one with trial two scores showed one subject to be markedly less consistent than the others ($p<.01$ by Grubb's test for detecting extreme values). This subject is excluded from the analysis presented here.

The design of the present study calls for an analysis of between subjects effects. Infants that receive one concentration of glucose solution on the 12 stimulations of each test trial are compared with infants receiving a different test fluid. Such comparisons are meaningful only to the extent that the infants are equated prior to testing. Although subjects brought to the criterion of adaptation are equated for level of responding, they may differ with respect to the number of stimulations they receive or the

* In accumulating the six successive failures to respond required to reach criterion on any trial, a single instance in which one but not the other observer reported seeing a response was accounted a failure to respond provided only that it was not the final or sixth failure to respond.

number of responses they made during adaptation. An analysis of variance performed on these two measures of adaptation indicates that groups based on age and test fluids do not differ significantly from each other and that trial one adaptation does not differ significantly from that of trial two. However, for each measure the main effect for sex is significant. Males require fewer stimulations of reach criterion and make fewer responses in the process. These findings must temper the interpretation of any sex difference in response to subsequent test stimuli.

Responding to the test fluids is a rising monotonic function of stimulus concentration. The percentages of test stimulation eliciting responses are 6.6, 12.0, 14.8 and 28.2 for water, 0.28, 0.55 and 2.77 M glucose, respectively. The data are more orderly for males than females (Figure 15–2). Separate analyses of variance (test fluids x ages x trials) for the number of observed responses yield significant effects for test fluids (Males; $F=7.6732$, $df=3/19$, $p< .005$: Females; $F=3.5231$, $df=3/18$, $p< .05$). The effects are uncomplicated by other significant main effects or interactions.

These data demonstrate that the newborn's tongue is differentially responsive to water and glucose solution when responsivity to water is first reduced to a low level by repeated stimulation. I will argue that they imply a receptor on the infant's tongue sensitive to some property that differentiates water from aqueous solutions of glucose and that this is a taste receptor.

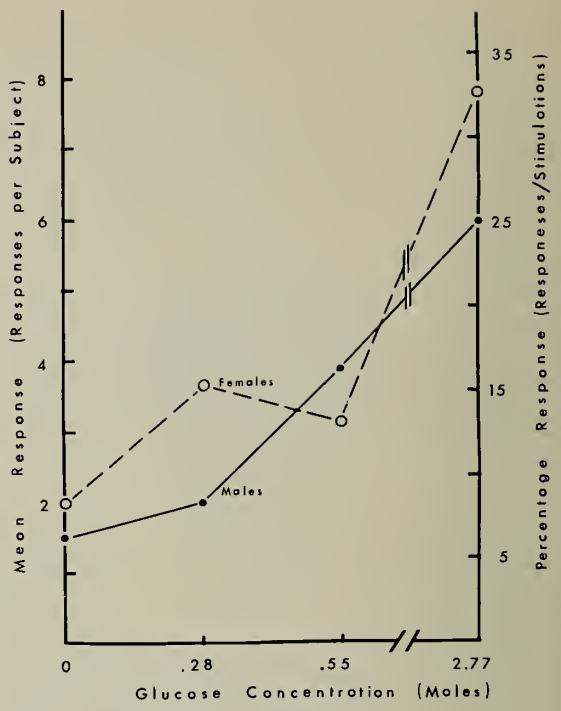


Figure 15–2. Independent groups of newborn infants were repeatedly stimulated with drops of water delivered to lateral sites on the dorsal surface of the tongue. When the subject's lateral tongue response to this stimulation reached a criterion low level (6 successive failures to respond) a 12 stimulus test was carried out with one of four test fluids, 2.77M, .55M, .28M glucose solution or water. The results of two repetitions of this procedure on the same day are presented graphically. Data are segregated by sex since females, although not significantly more responsive to initial stimulation with water, do require a significantly larger number of stimulations to reach the criterion of reduced responsivity. Separate analyses of variance for males and females each indicate a significant main effect for stimulus conditions. Neither analysis reveals significant main effects for age (6 to 30 and 30 to 72 hours) or repetitions. No interaction effects were significant. One female subject had a 1st to 2nd trial score differential markedly greater than any other subject ($p< .01$ by Grubb's test for detecting extreme values). This aberrant subject was excluded, reducing the number of subjects from 48 to 47.

INFERRING TASTE MECHANISMS

The role of adaptation

As has been pointed out previously,[5,7] an adaptation procedure such as that employed in the glucose demonstration would be unnecessary if test stimuli elicited differential levels of response before adaptation. In fact, the adaptation procedure would be unnecessary if repeated stimulation with test fluids elicited either differential initial levels of response or differential adaptation. Therefore, a comparison group of 9 male and 9 female newborns was drawn from the same population as the subjects of the glucose demonstration. These infants were treated just as the previous subjects except that they were initially stimulated with 2.77 M glucose. After responding to this fluid had been reduced to the previously employed criterion, twelve test stimulations with water were applied. Only one trial was administered. Neither the number of responses nor the number of stimulations required to reach criterion for drops of 2.77 M glucose solution differs significantly from that required when the adapting stimulus is water.[15] Thus, not only does the selective adaptation technique provide a demonstration of differential sensitivity to glucose solution and water but the adaptation to water drops is a critical feature of that demonstration.

The role of stimulus change

In the demonstration that glucose solution stimulation after adaptation to water drops elicits more responding than does continued water stimulation, the effects of increments in glucose concentration of the test fluid are confounded with possible effects of stimulus change *per se*. The additional infants initially stimulated with 2.77 M glucose solution and subsequently tested with water are exposed to a change in stimulus concentration equal in magnitude but opposite in direction to that provided to those subjects tested with 2.77 M glucose in the demonstration. Such infants fail to show the increment in response observed when the post-criterion change in concentration is in the reverse direction, from water to glucose. In fact, infants preadapted to glucose do not make significantly more test responses to water than infants receiving the water stimulation after adaptation to water.[15]

The direction of change in concentration between the adaptation fluid and the test fluid is critical. Increments, but not decrements in the concentration of glucose are associated with a resurgence of response. Thus, the mechanism involved appears not to be the familiar one of habituation [11] which has been so successful in studies of infant auditory and visual function (see ref. 7 for review). The mechanism appears, rather, to be one of sensory adaptation.

The role of response direction

The directionality of the lateral tongue movement response reduces the difficulty of discriminating a response from movements not elicited by the stimulus but occurring at the same time. Some might further suggest that the direction of the

response movement toward the stimulus reflects a positive hedonic evaluation of the eliciting stimulus. Perhaps the infant moves the tip of his tongue toward a drop of sugar solution because the drop is sweet and he likes (or at least approaches) sweet tasting things. The elicitation of similar ipsilateral tongue movements by touch or by drops of fluid that are not so obviously of positive hedonic value effects the argument very little. It is just such response as these that must be adapted by repeated elicitation with water drops before responses can be elicited whose frequency of occurrence is related to glucose concentration.

If responses to glucose solution after water adaptation are a function of hedonic tone, then hedonically negative fluids should either fail to produce response after adaptation or produce movements that are contralateral, away from the stimulus. Although, as Engen [3] has pointed out, it is difficult to find fluids that by their taste alone completely inhibit the newborn's intake, it is true that salt solutions have been shown to produce behavioral changes that are the reverse of those elicited by sugar solutions. Nowlis [10] has shown that salt solution may trigger responses at posterior tongue that can be considered complimentary to the acceptance movements of the anterior portions. In a study of infants less than 48 hours old, Aiyar and Agarwal [1] find salt solutions among those that, in contrast to sweet solutions, elicit a high frequency of crying, "dislike facies," vomiting and retching. Jensen,[9] making comparisons against a baseline established with milk, found saline produced a disruption or diminution of sucking, as a rising function of concentration. Crook has recently collected data suggesting that injecting minute quantities of saline into the infant's mouth during a pause in non-nutritive sucking prolongs that pause and reduces the length of the next burst, whereas sugar solutions similarly injected shorten the succeeding pause and increase the length of the next burst.*

Responses to drops of salt solution after adaptation to water thus are germane to the question of the role of hedonic tone in controlling the direction of the tongue movement response. Using the standard paradigm, twenty newborns (10 males and 10 females) were initially stimulated with water and subsequently tested with 2M sodium chloride (NaCl). Their post-adaptation response is compared with that of concurrently tested subjects that receive continued water stimulation after reaching criterion. The rate of responding to saline is approximately twice that for water (20.3% versus 10.3%). A three-way (test fluids x sexes x trials) analysis of variance of these preliminary data yield a significant main effect ($F=7.003$, $df=1, 32$, $p<.02$) reflecting the greater number of responses to saline. More importantly, for the present argument, these responses, like those elicited by glucose, are ipsilateral. Even these preliminary results are sufficient to reject the class of hypotheses that would attach hedonic meaning to the direction of the tongue movement response.

Defining receptor units

The elicitation of responses from

* Personal communication, Dr. Charles Crook, 13 March, 1974.

restricted sites on its surface allows for statements concerning the tongue's sensitivity to fluids. The selective adaptation paradigm permits further inference concerning mechanisms of reception.

The demonstration with glucose permits the definition of two receptor classes. One class of receptor unit is sensitive to some stimulus property shared by water and glucose solution drops. These receptor units may be operationally defined as water sensitive receptors. A more general characterization of them as units sensitive to "fluid touch" avoids the implication, which cannot be supported from the data at hand, that they are responsible for a water taste in human infants. The second class of receptor units that has been hypothesized is operationally defined by the sensitivity of such units to glucose. As I have pointed out elsewhere,[15] it is unclear whether units sensitive to glucose might not also be sensitive to other sugars, other sweet tastants, or even non-sweet tastants.

The evidence (see page 210) that the post-adaptation responding of infants adapted to water and tested with 2 M NaCl exceeded that of infants tested at the same time but receiving water throughout supports the inference that the infant's tongue is sensitive not only to glucose but also to NaCl. It is possible, of course, that the units which support responding to saline after water adaptation are the same as those that underly the response to glucose solution.

In an initial effort to clarify this point, twenty infants (10 males and 10 females) were adapted to 2M glucose solution. Half were then tested with 2M saline and half with 2M glucose. The infants tested with saline make more than twice as many post-adaptation responses (24.0% versus 10.0%). An analysis of these data by 3-way analysis of variance (test fluids x sexes x trials) indicates that the number of observed responses for saline after glucose adaptation is significantly greater than that for glucose after glucose adaptation ($F=7.873$, $df=1/16$, $p<.02$). These preliminary data are consistent with the hypothesis that separate peripheral mechanisms underlie tongue responses to saline and to glucose. They suggest, but do not demonstrate, that the receptors are differentially sensitive to taste quality.

SUMMARY

The laterally directed tongue movement response, serving as an indicator response in a selective adaptation paradigm, provides a measure of the sensitivity of the newborn's tongue to locatized chemical stimulation. A demonstration with glucose solution as the test stimulus suggests that the relation of the frequency of response occurrence to stimulus concentration is orderly. Other studies indicate that separate receptor mechanisms are involved in responses to drops of water, glucose solution and saline. Among the desirable characteristics of this technique are the separation of its indicator response from the normal sequence of feeding behavior and the control over stimulus parameters obtained by using minute amounts of stimulus fluid applied to discrete and de-

lineated sites on the tongue's surface.

To the degree that measures generated by this technique reflect peripheral sensitivity of tongue receptors rather than the complexly interacting mechanisms involved in the modulation of intake, they provide a unique point of contact with studies of taste reception in species where electrophysiological measures are available.

REFERENCES

1. Aiyar, R., and Agarwal, J.: Observations on the newborn: a study of 10,000 consecutive live births. *Indian Pediat.,* 6:729–742, 1969.
2. Collings, V.: Human taste response as a function of locus of stimulation on the tongue and soft palate. *Perception and Psychophysics,* 16(1): 169–174, 1974.
3. Engen, T.: The discrimination of dextrose and water by newborn infants (this volume).
4. Henkin, R.I., and Christiansen, R.L.: Taste localization on the tongue, palate, and pharynx of normal man. *J. Appl. Physiol.,* 22: 316–320, 1967.
5. Jeffrey, W.E., and Cohen, L.B.: Habituation in the human infant. In Reese, H.W. (Ed.). *Advances in Child Development and Behavior,* New York, Academic Press, 1971, pp. 63–97.
6. Jensen, K.: Differential reactions to taste and temperature stimuli in newborn infants. *Genetic Psychol. Monographs,* 12: 361–479, 1932.
7. Kessen, W., Haith, M.M. and Salapatek, P.H.: Human infancy: a bibliography and guide. In Mussen, P.H. (Ed) *Carmichael's Manual of Child Psychology,* (3rd ed.) New York, Wiley, 1970.
8. McBurney, D.H., and Pfaffmann, C.: Gustatory adaptation to saliva and sodium chloride. *J. Exp. Psychol.,* 65: 523–529, 1963.
9. Morrison, G.R., and Norrison, W.: Taste detection in the rat. *Candian J. Psychol.,* 20: 208–217, 1966.
10. Nowlis, G.: Taste elicited tongue movements in human newborn infants: an approach to palatability, In Bosma, J.F. (Ed.) *Fourth Symposium on Oral Sensation and Perception,* Washington, D.C., Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 292–310.
11. Sharpless, S., and Jasper, H.H.: Habituation of the arousal reaction. *Brain,* 79: 655–680, 1956.
12. Thach, B.T., and Weiffenbach, J.M.: Quantitative assessment of oral tactile sensitivity in premature and term neonates, and comparison with adults, *Develop. Med. Child Neurol.,* 18:204–212, 1976.
13. Weiffenbach, J.M.: Discrete elicited motions of the newborn's tongue. In Bosma, J.F. (Ed.) *Third Symposium on Oral Sensation and Perception,* Springfield, Thomas, 1972. pp. 347–361.
14. Weiffenbach, J.M.: Infants with clefts of lip and palate: observations of touch elicited oral behavior. In Bosma, J.F. (Ed.) *Third Symposium on Oral Sensation and Perception,* Springfield, Thomas, 1972, pp. 391–399.
15. Weiffenbach, J.M., and Thach, B.T.: Elicited tongue movements: touch and taste in the mouth of the neonate. In Bosma, J.F. (Ed.) *Fourth Symposium on Oral Sensation and Perception,* Washington, D.C., Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 232–243.

DISCUSSION

PFAFFMANN: First of all do you have a control for viscosity?

WEIFFENBACH: No, I don't yet have a control for viscosity. However, I believe that the studies I have described demonstrate a general paradigm that

can be used to determine to what extent the tongue is responsive to a wide variety of parameters of fluid stimulation including viscosity.

PFAFFMANN: Well, I agree with you entirely about the methodology. In fact, if you read Schwartz's paper on pheromones in the sugar glider which has a very elaborate olfactory communication system you'll see that the way he proves the generality of the different pheromones glands is essentially the method you are using.

WEIFFENBACH: Yes, after struggling to describe my procedures, I was chagrined to find Deither had described an essentially similar technique with striking clarity in a half dozen lines.[1]

PFAFFMANN: Have you considered using bilateral stimulations simultaneously with different combinations of sugar solutions and water at two sites on the tongue following the model that von Bekesy developed?

WEIFFENBACH: Yes, I think it would be difficult but interesting to do. At this time I have no evidence that bilateral equal stimulation will summate to cancel response. However, at an extremely crude level there is some indication that unequal bilateral stimulation will lead to directional response. If one side of the tongue is adapted to fluid drops, then a drop placed at the center line of the tongue is likely to elicit a movement toward the side that has not been adapted. The stimulus drop is physically on the center line but due to adaptation the effective stimulation is bilaterally unequal. More importantly, perhaps, it would appear that the effects may be independent of where on the adapted side the stimulus has been applied.

To return to the question of bilateral simultaneous stimulation it is interesting to me that bilateral simultaneous touch stimulation of the arms show a developmental course. Perhaps this is a good technique for exploring growth related changes in the nervous system.

PFAFFMANN: You get an exaggeration in the brain-damaged patient so that he feels only one side. So there is a suppression.

What about the water taste? You would expect that the water after glucose would taste bad.

WEIFFENBACH: It may be that it does but I don't know how I can demonstrate that it does. My data would suggest that applying water after glucose solution stimulation has adapted the glucose sensitive tongue receptors is much like continuing to stimulate with the glucose solution since glucose sensitive receptors are nonresponsive. For the measures I am using it makes little difference if one functionally suppresses the glucose sensitive receptor or physically removes the glucose from the stimulating fluid.

---

[1] Dethier, V.G. and Goldrick, N.: Blowflies: Alterations of adult taste response by chemicals present during development. *Science*, 173:242–244, 1971.

PART IV
TASTE EXPERIENCE AND THE DEVELOPMENT OF A DIETARY PREFERENCE FOR SWEET

Chapter 16

MECHANISMS FOR THE TRANSMISSION OF ACQUIRED PATTERNS OF FEEDING FROM ADULT TO WEANLING RATS

Bennett G. Galef, Jr.

There are, broadly speaking, essentially three nonindependent mechanisms whereby the behavior characteristic of a population may be maintained from one generation to the next. First, adaptive behavior in population members may be largely endogenously organized and genetically transmitted as propensities guiding development. Second, similar patterns of behavior in population members may result from similar reinforcement histories of individuals in their direct transactions with the nonsocial environment. And, third, long term homogeneity of population behavior may result from the social transmission of patterns of behavior from individual to individual within a population. Both the rejection of the Lamarkian theory of the genetic transmission of acquired characteristics and the difficulty of adequately demonstrating learning by observation in animals [13] have served to minimize the role accorded the social transmission of acquired patterns of behavior in behavioral development. Therefore, in discussing the mechanisms responsible for the social transmission of acquired feeding preferences in rats, I hope to direct attention generally to an aspect of the ontogeny of behavior which has been largely ignored in recent years.

WEANING AND POISON AVOIDANCE IN RATS

The period during which altricial mammals wean from a milk diet, to one of solid food is a time of stress and major mortality in many mammalian species. Unless adult conspecifics transport requisite solid food to the nest site, the young must find it for themselves in an unfamiliar and potentially hostile environment in which they are particularly vulnerable. Clearly, it would be to the advantage of the young and hence to the reproductive advantage of their parents if adults could, in some way, make avail-

Acknowledgment: The author would like to thank the Smithsonian Tropical Research Institute for providing facilities and the National Research Council of Canada for providing financial support during the drafting of this chapter. Thanks are also due to Lorraine Allan, Mertice Clark, and Michael Leon for their thoughtful criticisms of an earlier draft.

able to their offspring information concerning the location and safety of the various foodstuffs available in the vicinity of the homesite. This is information which the adults have acquired during their own lives in the area in which they rear their young. The problems of diet location and selection may be particularly acute for wild rats, which are commensals of man,[23] as they are liable to encounter poison baits in areas in which they are sympatric with mankind. Norway rats (*R. norvegicus*) have evolved a variety of behavioral mechanisms permitting individuals to avoid ingesting lethal amounts of poisons [1,12,17,18,19,20,21] and it is at least possible that they have also found ways to communicate information to one another regarding the safety and location of various foods in the environment.

EVIDENCE OF SOCIAL TRANSMISSION OF FEEDING PREFERENCES

Steiniger,[22] in discussing the "local traditions" of colonies of wild Norway rats (i.e., consistent behavioral differences between groups of rats living in different areas) has reported that if a given poison bait is used in one area over a considerable period of time, despite initial success, later acceptance of that bait falls off radically and remains low. He noted that the offspring of surviving animals rejected the bait so long as a few animals in their pack knew and rejected it. Steiniger attributed this "traditional" poison avoidance to the behavior of experienced animals which in some way dissuaded the naive young from ingesting poison baits.

During the past several years, my students and I have been examining the social interactions of both wild and domesticated rats during weaning under controlled conditions. We have worked within the general context of poison avoidance behavior seeking possible mechanisms for the social transmission of acquired feeding preferences.

In our basic experimental paradigm [6] colonies consisting of two male and four female wild rats are established in enclosures like the one diagrammed in Figure 16–1a. Water is continuously available and food is presented to the colony for 3 hr/day in two ceramic food bowls located approximately 2½ ft. apart. Each of these bowls contains one of two nutritionally adequate diets, each discriminable from the other in color, texture, taste and smell. These two diets will be referred to below as Diets A and B. Diet A is powdered Purina Laboratory Chow and Diet B, compounded by the General Biochemicals Company, consists mainly of sucrose and casein and is highly preferred by rats to Diet A.

The adult members of the colony are trained to eat one diet and to avoid the other, by introducing sublethal doses of poison (lithium chloride) into the samples of one of the diets offered to the colony during daily 3-hr. feeding periods. Under these conditions, our wild rats rapidly learn to avoid ingesting the poisoned diet and continue to avoid it for some weeks even when subsequently offered the once poisoned diet free from lithium chloride contamination.

The experiment proper begins when litters of pups born to colony members (reduced to 6 pups/litter)

leave their nest site to feed on solid food for the first time. We observe the adults and pups throughout daily 3-hr. feeding periods via closed circuit television and record the number of times the pups approach to within 4 in. of each food bowl and the number of times they eat from each food bowl.

After the pups have been feeding on solid food for a number of days, they are transferrred to new enclosures, (illustrated in Figure 16–1b) where, without the adults of their colony, they are again offered the choice

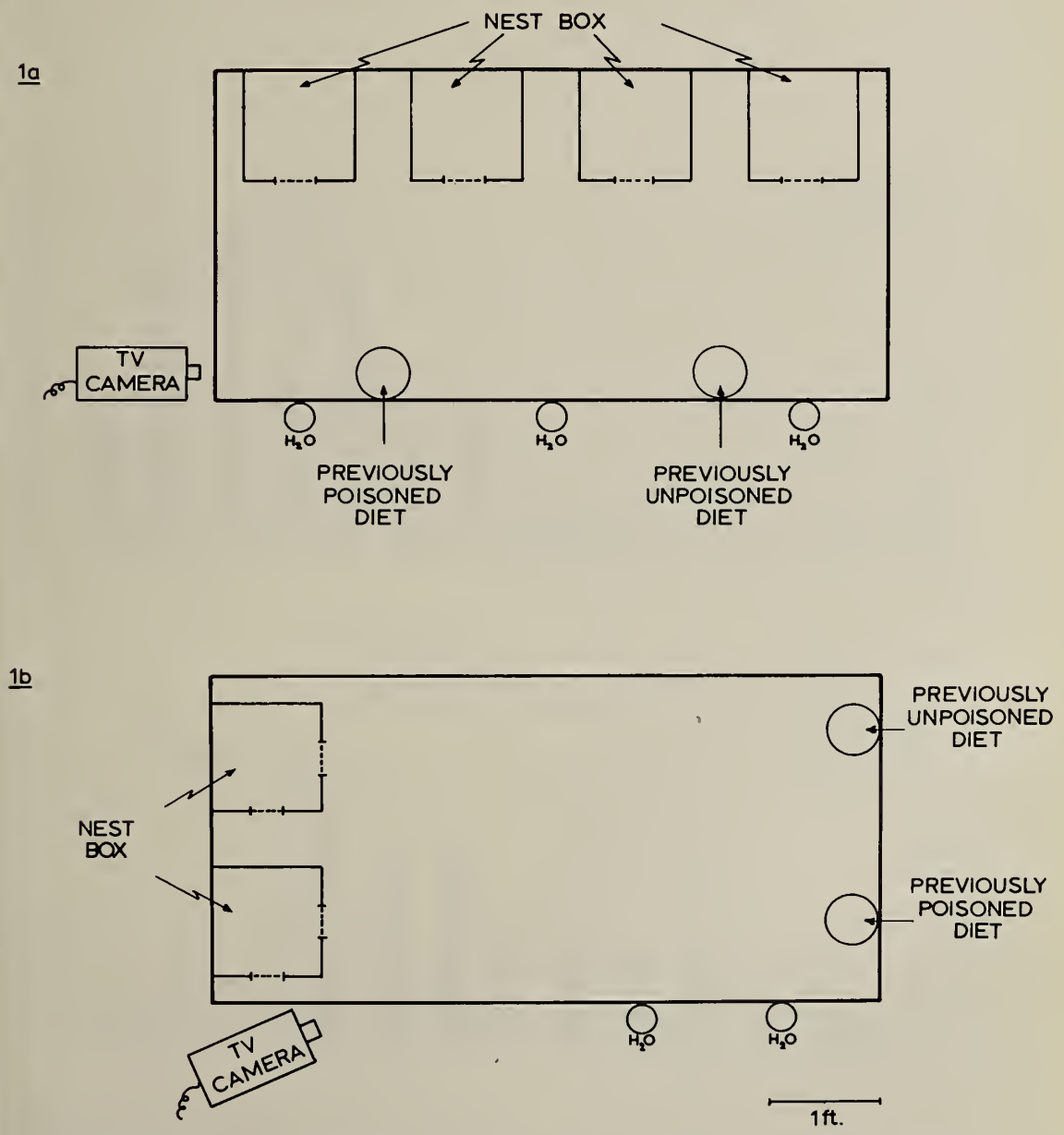


Figure 16–1. Enclosures in which rat pups were observed prior to (a) and after (b) removal from their adult clan.

of uncontaminated samples of Diets A and B for 3 hr/day. The amount of each diet eaten by the pups in this situation is determined by weighing food bowls before and after each feeding session.

The results are presented in Figures 16–2a, 2b, and 3. Figure 16–2a presents data on the feeding behavior of a litter of wild rat pups born to a colony previously trained to avoid ingesting the normally preferred Diet

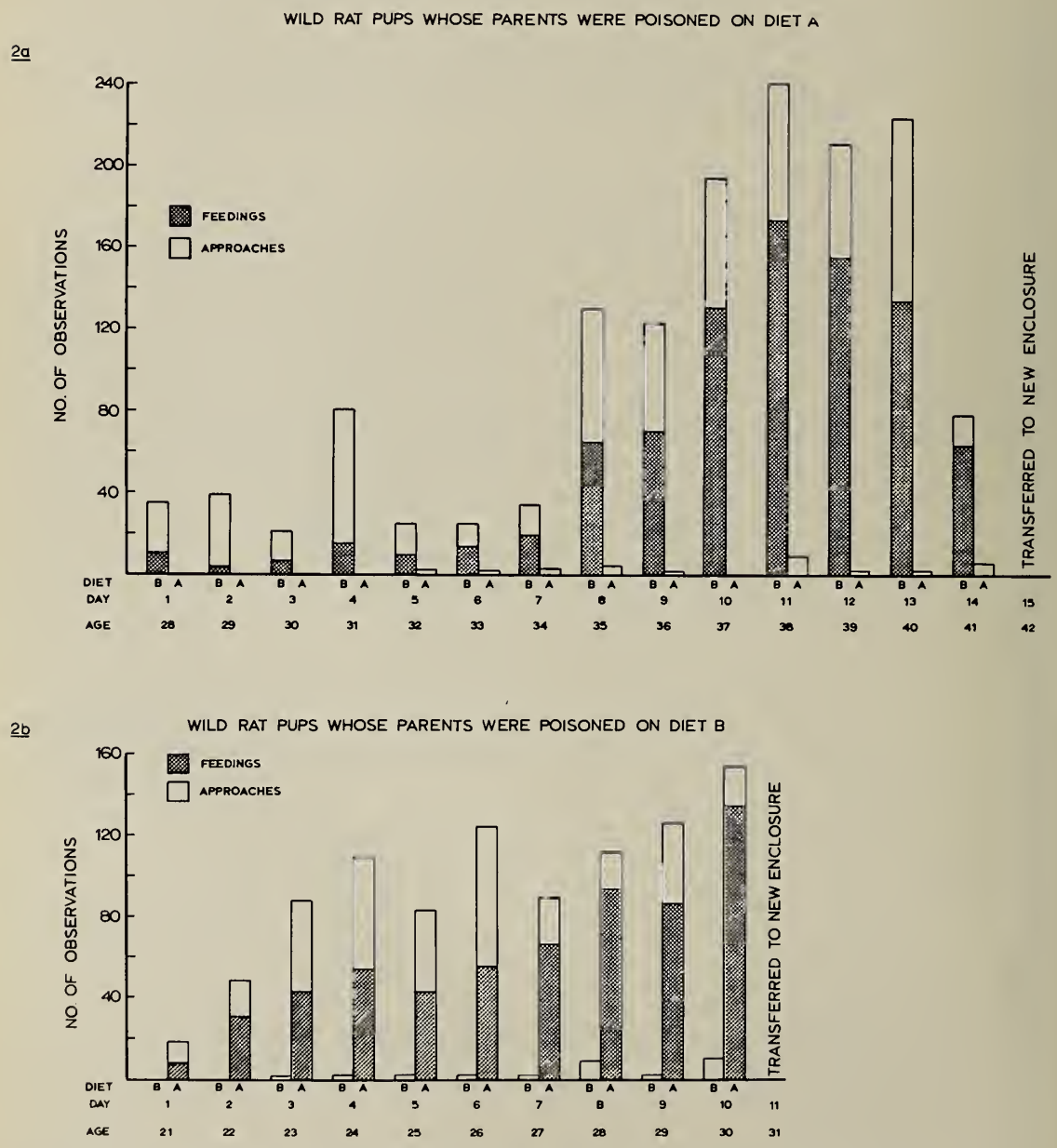


Figure 16–2. Number of observed approaches to and feedings from Diets A and B by wild rat pups the adults of whose colonies had been poisoned on Diets A (a) and B (b).

B, while Firgure 16–2b presents data describing the feeding behavior of a litter of pups born to a colony trained to avoid ingesting Diet A. It is clear from comparison of the data presented in Figures 16–2a and 2b that the learned feeding preferences of adult colony members profoundly affect the feeding preferences of their young. In the presence of adults of their colony, pups ingest only the diet that the adults are eating. Furthermore, as shown in Figure 16–3, the dietary preferences of the adults continue to affect the feeding preferences of their young for some 8 to 10 days following transfer of the pups to enclosures separate from the adult colony.

Taken together these observations demonstrate that adult rats can, in some fashion, lead their offspring to feed solely on a safe diet in an environment containing food known by the adults to have been poisoned,

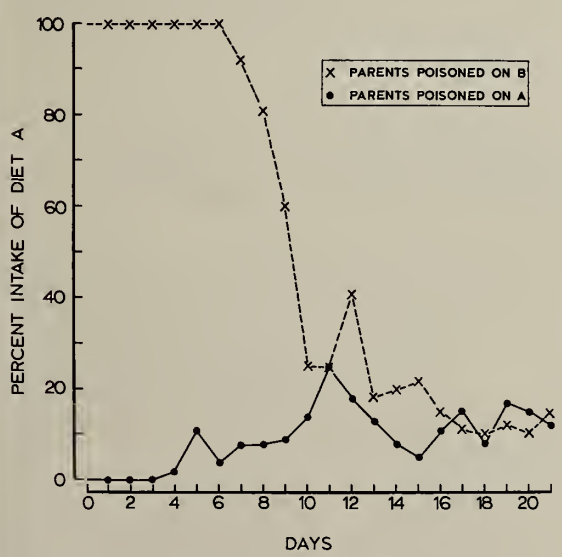


Figure 16–3. Percentage intake of Diet A by pups following removal to the enclosure illustrated in Figure 16–1(b) isolated from adults.

thereby allowing the young to avoid ingesting potentially noxious foods. The data also show that the food preference learned in the presence of the adults continues to affect the feeding behavior of the pups for some time following their removal from direct adult influence.

It is not possible to determine from these data whether the pups learn to ingest the diet which the adults of their colony eat or to avoid ingesting the diet which the adults of their colony avoid. It is, however, important to determine the precise nature of the behavior being transmitted from adults to young before attempting an analysis of underlying mechanisms.

If pups learn to avoid the diet which the adults of their colony avoid as a result of interaction with them, then it should be important that the pups experience that diet during the time they are with the adults. Conversely, if the pups learn only to eat what the adults of their colony eat, then the presence of the adult-avoided diet during the period of adult-young interaction should be of no importance.

The experimental design used to investigate this question was very similar to the one just described. Colonies of adults were trained to avoid the normally preferred Diet B and to eat Diet A. Pups were allowed to feed with the adults for 10 days and were then moved to individual enclosures (see Figure 16–4) and offered the choice of uncontaminated samples of Diets A and B. While in the enclosure with the adults for 10 days of feeding, pups of one colony and their adults were offered the choice of Diets A and B (and ate only Diet A) while pups and adults in another colony

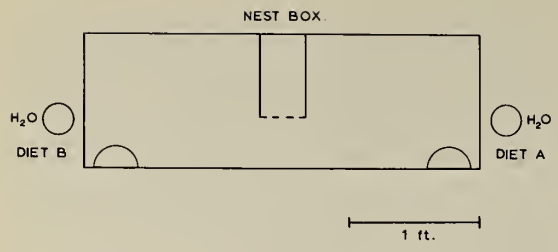


Figure 16–4. Enclosure to which individual pups were removed following 10 days of feeding with adults.

were offered the choice between Diet A and an empty bowl placed in the location in which Diet B was usually available. If pups in the original experiment learned to avoid the diet that the adults of their colony avoided, then one would expect pups which had Diet B available while with the adults to show a more profound avoidance of Diet B, when offered the choice of Diets A and B in individual enclosures, than pups which had not previously seen Diet B.

Figure 16–5 illustrates the percentage of Diet A eaten by pups in the two groups when offered the choice of

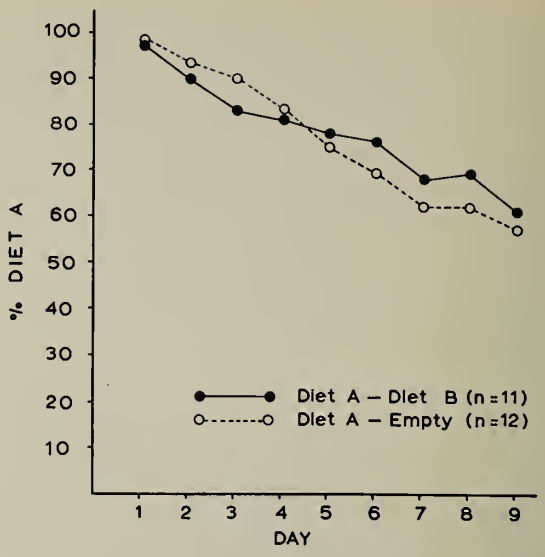


Figure 16–5. Amount of Diet A eaten by individual pups while in the enclosures illustrated in Figure 16–4.

Diets A and B in individual enclosures. As is clear from examination of the figure, there is no major difference between the two groups. The simplest interpretation of this data is that pups learn to eat the diet that the adults eat rather than to avoid the diet the adults avoid.

MECHANISMS FOR SOCIAL TRANSMISSION OF FEEDING PREFERENCES

Adult presence at a feeding site

Any of a wide range of mechanisms might be responsible for this transfer of an acquired feeding preference from adult rats to pups. For example:

(1) Adults may mark either the diet they are eating or the diet they are avoiding with residual chemical cues which attract or repel the pups and thereby influence their feeding site selection.

(2) Adults may carry samples of the diet they ingest back to the nest site and ingestion of these samples may influence pups' subsequent choice of diet.

(3) The physical presence of adults at a feeding site may attract pups to that site and influence their choice of diet for early ingestion.

If adults deposit attractive chemicals in or near the food they are eating or if they mark the food they are avoiding or its surrounding area with some warning chemical, and these residual chemicals are responsible for adult influence on pups' initial choice of diet, then it should be fairly easy to disrupt the apparent influence of the adults on the food choices of their young. All that would be needed would be to disturb the chemicals deposited by the adults. We have conducted the following experiment in order to determine the importance of such chemical cues for the behavior of the pups. The experimental situation was basically the same as that of the first experiment I described except that sheet metal trays (2 x 2 ft.) covered with sawdust were placed under both food bowls during both the training of the adults and the testing of the pups. When pups in each of the litters we used had been observed to eat solid food 20 times, we performed the following manipulations: (1) The two food bowls were emptied. (2) They and the trays and sawdust the food bowls were sitting on were reversed in position, and (3) new samples of Diets A and B were placed in their initial positions in the now reversed food bowls. Thus all chemical cues located within 1 ft. of the food bowls had been reversed in position within the experimental enclosures.

These manipulations had absolutely no effect on the feeding behavior of the pups.[6] Without exception, the pups continued to eat Diet A and avoid Diet B, as did the adults themselves. These data indicate that the deposition of residual chemical cues is not necessary for transmission of the feeding preference. I will discuss the question of whether chemical cues are sufficient to determine pups' feeding preferences later.

To look at the effects of the ingestion of food stuffs at the nest site on subsequent food choices, one can offer the pups in the nest site, at some time other than the 3–hr. colony feeding period, samples of the food the adults of their colony have been trained to avoid and then examine the pups' food preferences during the colony feeding periods.[2] The results of such an experiment are presented in Figure 16–6. There are two interesting aspects of the data. First, sampling by the pups in the nest site of the adult-avoided diet did not produce an *initial* preference for that diet. Second, such sampling did affect the later rate of acceptance by pups of the adult-avoided diet. I will be returning to the second point later.

If neither residual chemical cues at a feeding site nor ingestion of food samples in the nest site determined initial dietary preference in our first experiment, what did? The evidence indicates that the most important factor influencing pups' choice of initial diet is the presence of adults at a food site at the time when pups are ready to begin feeding on solid food. In illustration of this point, consider the following experiment.[7] A colony of adult wild rats was established in the enclosure illustrated in Figure 16–7. Diet A was constantly available in the two food bowls located behind the partition and the area above the dotted line was continuously monitored via closed circuit television. We marked pups individually and looked to see under what conditions each pup ingested its first meal of solid food. We observed 9 pups in all and

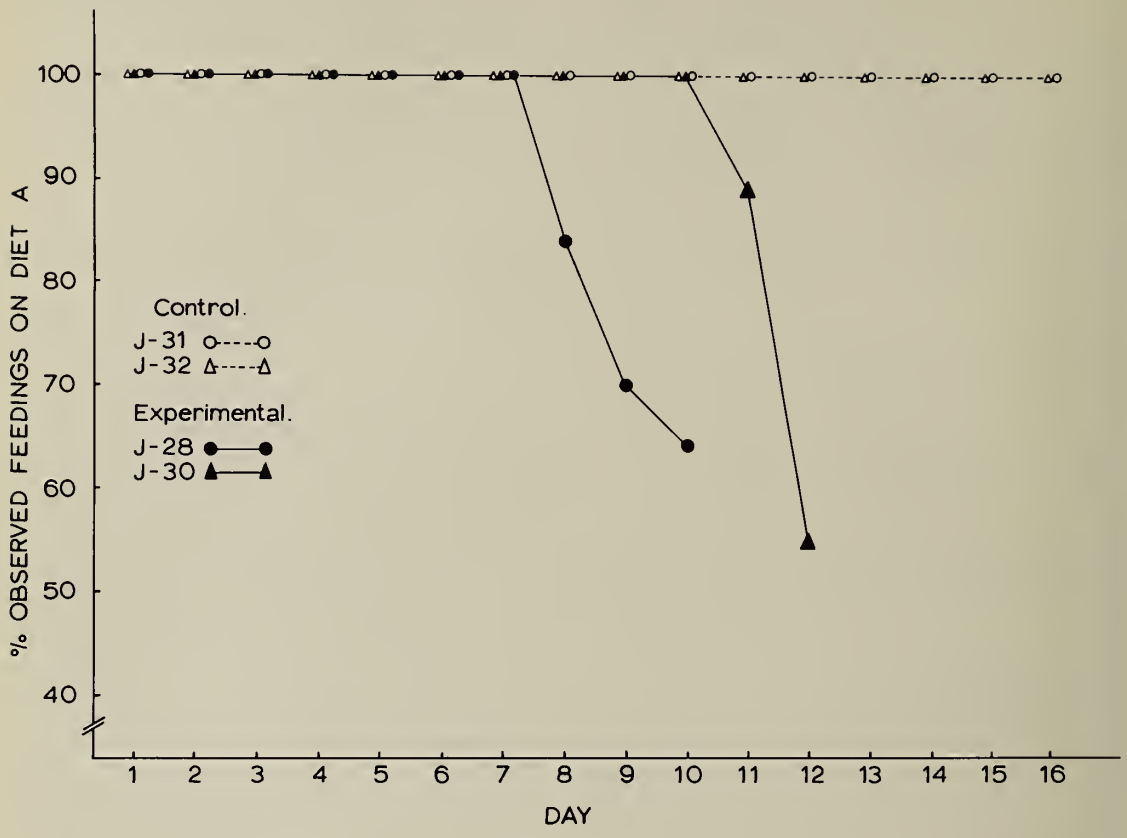


Figure 16–6. Percentage of observed feeding responses directed to Diet A during the colony feeding period by litters of pups offered samples of the diet the adults of their colony were eating (control) and samples of the diet the adults of their colony were avoiding (experimental) in the nest site.

all 9 of them ate their first meal under exactly the same circumstances. All 9 ate their first meal in the presence of a feeding adult even though the probability of that occurring by chance, given the temporal distribution of adult meals, was less than .001, and all 9 ate at the same food bowl as that adult and not at the other bowl some 1½ ft. away. The presence of an adult at a feeding site seems to attract pups to that site and cause pups to initiate feeding on the diet present there. Our further observations and experiments within this enclosure indicate that the pups tend to approach adults outside the nest area rather than to follow them from the nest site to food, and that visual cues are necessary to guide this approach. Blinded pups showed no tendency to eat their first meal of solid food in the presence of a feeding adult.[7]

So one way in which adults can influence the initial food choices of pups during weaning is as a result of their physical presence at a feeding site. The entire phenomenon, early total avoidance of the adult-avoided diet, followed by continued avoidance

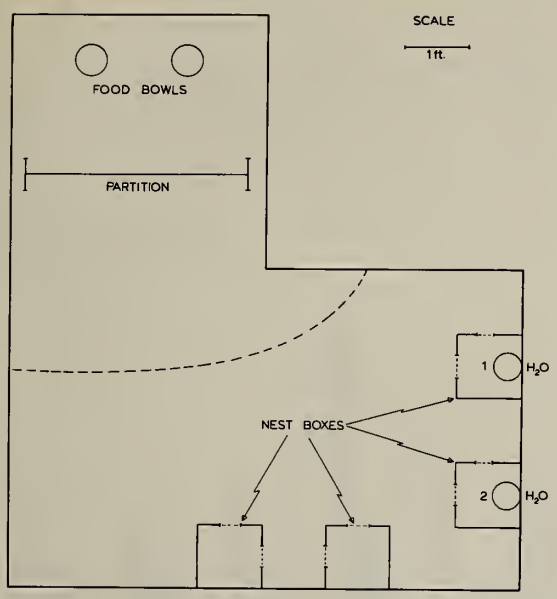


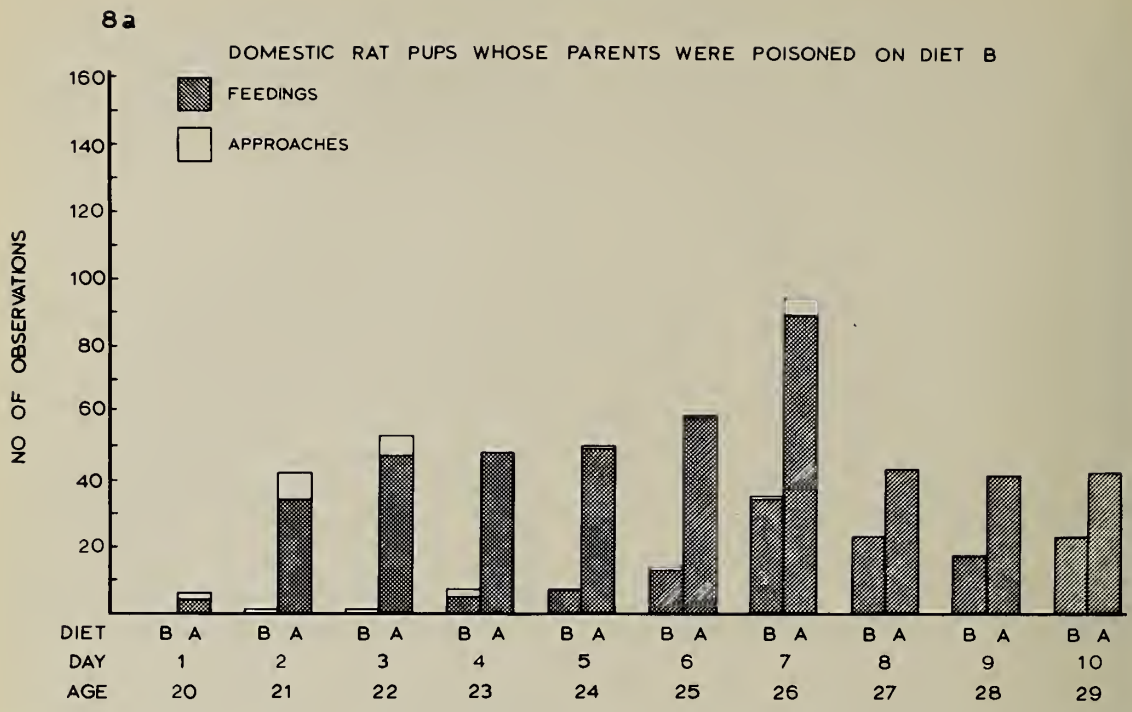
Figure 16–7. Enclosure for wild rat colony.

in isolation, followed by gradual acceptance of the previously avoided diet can, perhaps, be explained in terms of a three-stage process. Initially, the pups tend to approach adults at a distance from the nest site and begin feeding in their vicinity. They become familiar with the diet which the adults are eating, and then avoid alternative diets because of their novelty.

Wild rats have been described as strongly neophobic,[1, 3] that is, they show a strong tendency to avoid novel foods or objects in the environment. This neophobia could be responsible for continued avoidance of alternative diets once the pups have become familiar with the diet which the adults of their colony are eating. You will remember, for example, that pups who had become familiar with the adult-avoided diet in the nest site, began feeding on that diet sooner than controls who had not been exposed to the adult-avoided diet (Fig. 16–6). If, in fact, neophobia is at least in part responsible for continued avoidance, one would expect domestic rat pups, which are at best only mildly neophobic,[1, 18] to behave quite differently from wild pups in our basic experiment. They should initially follow adults to the diet they were trained to eat,[4] but should then transfer feeding to the adult-avoided alternative more rapidly than wild rats. Figures 16–8a and 8b report observations on two litters of domestic pups observed in our basic experiment. The data support the notion that neophobia is important in maintaining the pups' avoidance of the adult-avoided diet. Domestic rat pups, although initially feeding on the same diet as the adults of their colony, begin to eat the adult-avoided alternative after some 3 to 5 days.

Flavor cues in mothers' milk

We have conducted an experiment [8] rather similar to the first one I described, but different in one very important respect. Colonies of domestic rats were again housed in 3 x 6 ft. enclosures. However, in this experiment the adults were removed to a separate cage where they were fed either Diet A or Diet B for 3 hr/day depending on the experimental condition to which their colony was assigned. While the adults were out of the colony enclosure, the pups were presented with two standard food bowls, one containing Diet A and the other Diet B. We weighed the food bowls after each 3-hr. feeding session to determine the amount of each diet eaten by the pups. The results are pre-


8a
DOMESTIC RAT PUPS WHOSE PARENTS WERE POISONED ON DIET B
FEEDINGS
APPROACHES
NO OF OBSERVATIONS
160
140
120
100
80
60
40
20
DIET B A B A B A B A B A B A B A B A B A B A
DAY 1 2 3 4 5 6 7 8 9 10
AGE 20 21 22 23 24 25 26 27 28 29

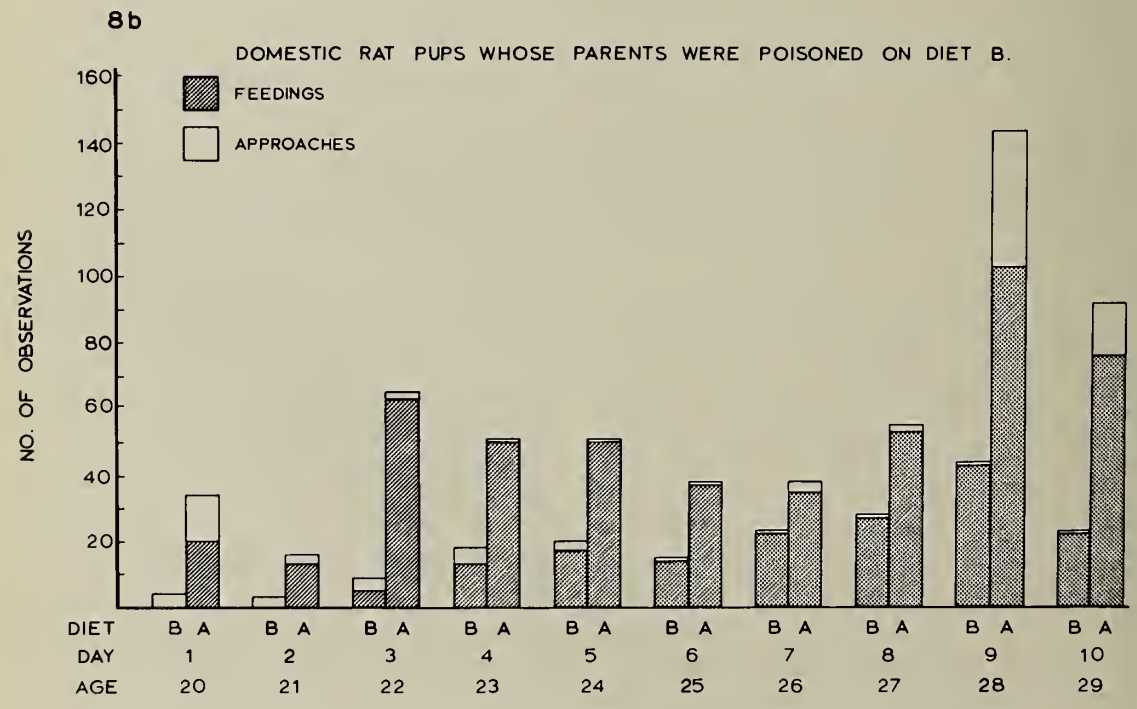

8b
DOMESTIC RAT PUPS WHOSE PARENTS WERE POISONED ON DIET B.
FEEDINGS
APPROACHES
NO. OF OBSERVATIONS
160
140
120
100
80
60
40
20
DIET B A B A B A B A B A B A B A B A B A B A
DAY 1 2 3 4 5 6 7 8 9 10
AGE 20 21 22 23 24 25 26 27 28 29

Figure 16–8. Number of observed approaches to and feedings from Diets A and B by domestic rat pups the adults of whose colony had been poisoned on Diet B.

sented in Figure 16–9. As is apparent from examination of the figure, the diet being fed to the adults is affecting the food choices of the pups even though, under these conditions, the pups and adults have no opportunity to interact directly in the feeding situation.

Again there are a number of possible explanations:

(1) The flavor of the diet ingested by an adult rat may directly affect the flavor of its feces and ingestion of adult feces may cause pups to preferentially ingest diets of a similar flavor during weaning.

(2) Particles of food might cling to the adults' fur and vibrissae, and the pups could ingest these and become familiar with the parents' diet.

(3) The flavor of the diet ingested by a lactating female rat during the nursing period might directly affect the flavor of her milk and cause pups to preferentially ingest similar tasting diets during weaning.

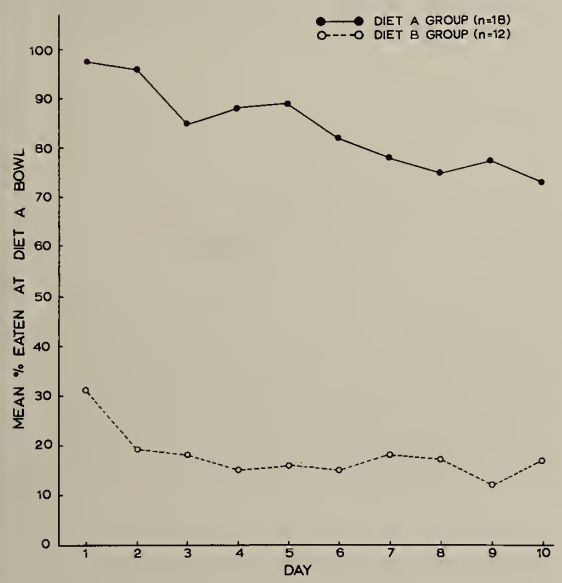


Figure 16–9. Percentage of Diet A eaten by pups the adults of whose colony are eating Diet A (Diet A group) and Diet B (Diet B group) when adults and pups have no opportunity to interact in a feeding situation.

Our data indicate that it is the third alternative which is the most likely one.[10] That is, the milk of a lactating female rat contains gustatory cues reflecting the taste of her diet with sufficient accuracy to enable her pups to identify that diet during weaning. Let me describe an experiment which supports this conclusion.[11] Rat pups nursing from a lactating conspecific female eating Diet A, were force-fed ½ cc. of milk hand expressed from a second lactating female eating Diet B and then poisoned by intraperitoneal injection of lithium chloride following their force-fed meal. If pups treated in this way show an aversion during weaning to Diet B itself, the diet ingested by the female from whom milk was hand expressed, in comparison with controls, then transfer of gustatory cues reflecting a lactating female's diet would be clearly demonstrated. Figure 16–10 presents relevant data.

The figure indicates the amount of Diet B eaten, as a percentage of total intake, by pups during their first 24 hr. of feeding on solid food. Pups in all groups were reared by mothers eating Diet A. Those in the experimental group received a force-fed meal of milk from a lactating female eating Diet B before being injected with a toxin (lithium chloride), while those in the control groups were injected with toxin 24 hr. prior to receiving milk from a lactating female eating Diet B (temporal control), force-fed milk from a lactating female eating Diet A immediately before

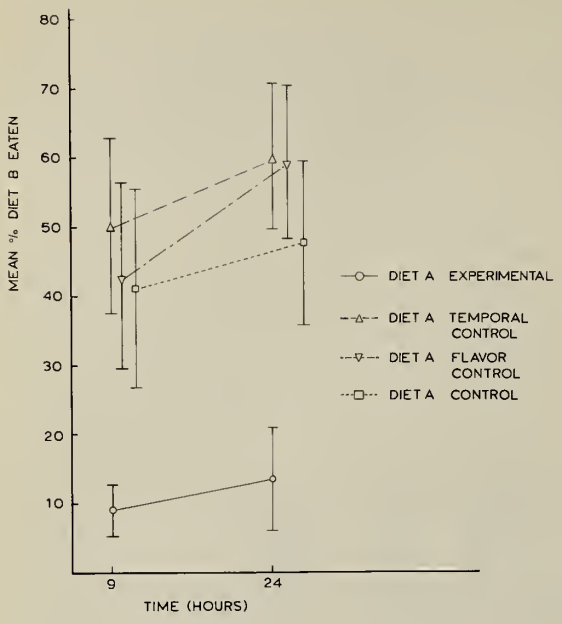


Figure 16–10. Mean percentage of Diet B eaten by pups during their first 24 hr. of feeding on solid food. (Points are displaced laterally to facilitate reading of the figure. Flags indicate ± $1SE_M$.)

toxin injection (flavor control), or force-fed milk from a lactating female eating Diet A but not injected (control). Clearly, only those subjects poisoned immediately following ingestion of milk from a female eating Diet B show a strong aversion to Diet B at weaning.

At this point one might reasonably ask if the results of the first series of experiments, which were explained in terms of the influence of adult presence could not also be explained solely in terms of cues contained in mother's milk. We have conducted an experiment which indicates that adult presence at a feeding site is sufficient to determine a pup's choice of location for early ingestion independent of cues contained in mother's milk.[8] A colony of adult wild rats was offered Diet A for 3 hr/day in two metal bowls located about 2½ ft. apart. We trained the adults to feed at one location by shocking them whenever they ate at the other. Diet A was present in both food bowls and, therefore, cues contained in mother's milk would be of no use to pups in selecting one feeding site over another. Thus, the results provide a test of whether adult presence is sufficient to determine feeding site selection in a situation that is not confounded by the influence of mother's milk. As is apparent from examination of Figure 16–11, which indicates the percentage of feedings by pups at the location at which adults fed, adult presence has a profound effect on the feeding site preferences of the young independent of the effects of cues incorporated in mother's milk. The comparatively rapid transition by the pups to feeding at the site avoided by the adults supports the interpretation of maintenance of avoidance as depen-

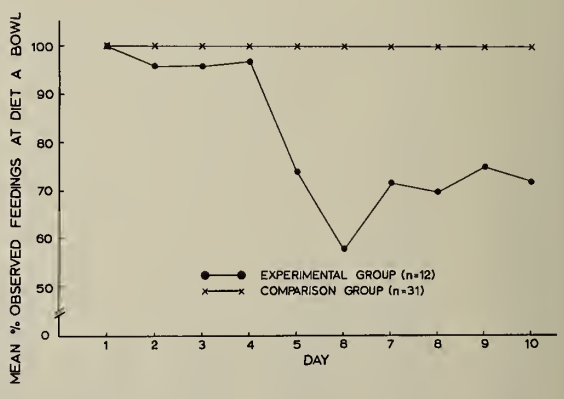


Figure 16–11. Mean percentage of feedings by wild rat pups at the food bowl from which the adults of their colony were eating (Diet A Bowl) when Diet A was present in both food bowls (experimental group) and when Diet A was present in one food bowl and Diet B in the other (comparison group).

dent on avoidance of novel foods.

There are, then, at least two mechanisms, each sufficient to allow adult rats to influence their youngs' choice of diet for initial ingestion. First, gustatory cues incorporated in a lactating famale's milk reflecting the flavor of her diet, are sufficient to allow pups to recognize that diet and to cause pups to preferentially ingest it at weaning. Second, the physical presence of adult rats at a feeding site attracts pups to that site and, consequently, can affect their choice of diet for early ingestion.

Residual chemical cues

As a result of consideration of data from one of our earlier experiments, it was concluded that chemical cues were not necessary for the adults to influence the feeding preferences of pups. More recently Leon and Moltz [14, 15, 16] have demonstrated that lactating female rats excrete material which has an odor highly attractive to rat pups from 16 to 23 days of age. Consideration of this data led us to examine in greater detail the role of residual chemical cues in the determination of feeding site preferences in weanling rats. Even though our data indicate that chemical cues are not necessary for the adults to direct pups to a specific feeding location, it is possible that they are sufficient for that purpose. The experiment described below was designed to determine whether or not chemical cues deposited in an environment are sufficient to determine pups' choice of location for early ingestion of solid food.[9]

A lactating female rat and her litter were established in the enclosure diagrammed in Figure 16–12. The female and litter were left in the enclosure 24 hr/day on ad lib food and water until the pups reached 17 days of age. When the litter reached 17 days of age, they and their dam were removed from the enclosure for 3 hr/day for seven consecutive days. During the period when the famale and litter were absent from the enclosure, pups of the same age, taken from litters reared without the opportunity to ingest solid food, were placed individually in the enclosure and the barrier illustrated in Figure 16–12 was removed. Each pup was observed on closed circuit television for 50 min/day for seven consecutive days. The amount of time each pup spent eating from each food bowl and the amount of time each pup spent exploring in the previously occupied and unoccupied end thirds of the enclosure were recorded. The data are presented in Figure 16–13.

It is clear that chemical cues deposited by a lactating female rat are sufficient to markedly affect the feeding locations selected by pups during weaning.

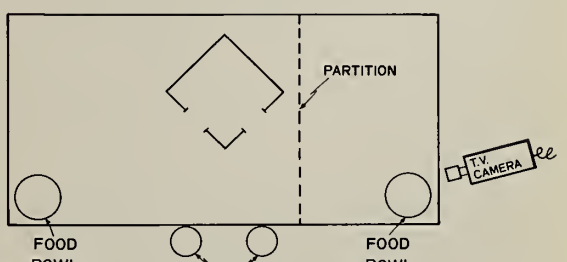


Figure 16–12. Enclosure used to determine the effects of chemical cues on the exploratory and feeding behavior of domestic rat pups.

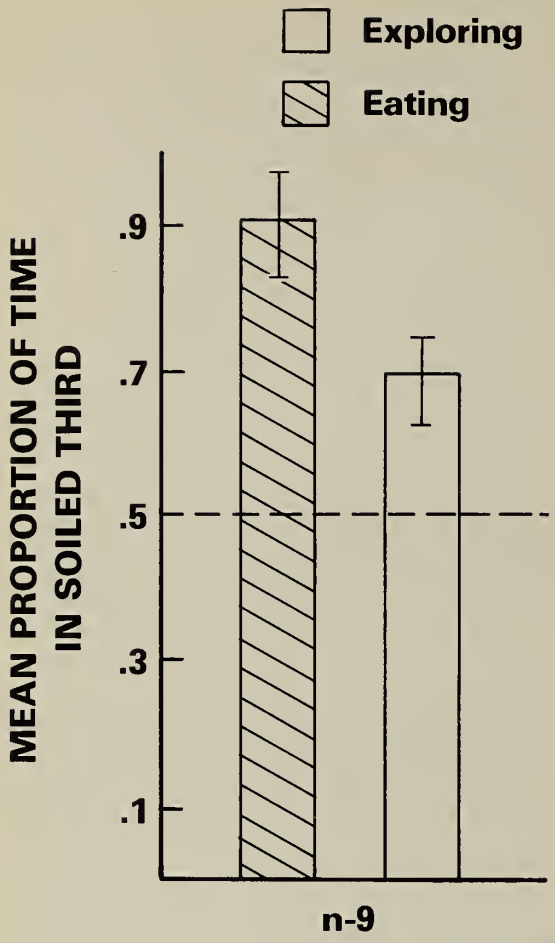


Figure 16–13. Mean proportion of time spent by individual pups exploring and eating in the end of the enclosure previously occupied by a lactating female and her litter.

CONCLUSIONS

It would appear, then, that adult rats can affect their pups' choice of diet for initial ingestion in three ways: via mother's milk, via their physical presence, and via the deposition of residual chemical cues. Each of these three mechanisms is sufficient and none necessary to support the social transmission of an acquired feeding preference.

Both consideration of the data presented above and review of the literature suggest that the social transmission of behavior results from a combination of the effects of a social interaction between transmitter and recipient, and a predisposition in the recipient to respond in a particular way to stimuli encountered as a result of that social interaction.[5] The transmitter can, for example, alter the environment in such a way as to channel or direct the behavior of the recipient. In the present series of experiments environmental alteration was achieved via the physical presence of adults at a feeding site and by the deposition of chemical cues. Alternatively, responses of the recipient directed toward the transmitter can introduce the recipient to selected aspects of the environment to which the recipient then responds in a specific fashion. The use of flavor

cues incorporated into mother's milk is such a process, as is the visually directed approach of pups to adults.

The evidence strongly suggests that the social transmission of acquired behavior is an important element in the adaptation of rats to their environment. Rats have evolved behavior patterns which permit weanlings to incorporate the acquired feeding preferences of adults of their colony into their own behavioral repertoires. This transmission of acquired feeding patterns should greatly facilitate the pups' transfer of feeding from milk to solid food, reducing the time spent by young rats in locating needed foods in the environment, and reducing the probability that they will ingest poison foods.

REFERENCES

1. Barnett, S.A.: Experiments on "neophobia" in wild and laboratory rats. *British Journal of Psychology*, 49:195–201, 1958.
2. Clark, M.M. and Galef, B.G., Jr.: The effects of forced nest site feeding on the food preferences of wild rat pups at weaning. *Psychonomic Science*, 28:173–175, 1972.
3. Galef, B.G., Jr.: Aggression and timidity: Responses to novelty in feral Norway rats. *Journal of Comparative and Physiological Psychology*, 70:370–381, 1970.
4. Galef, B.G., Jr.: Social effects in the weaning of domestic rat pups. *Journal of Comparative and Physiological Psychology*, 75:358–362, 1971.
5. Galef, B.G., Jr.: Social transmission of acquired behavior: A discussion of tradition and social learning in vertebrates. *Advances in the Study of Behavior*, Vol. 6. New York, Academic Press, In press.
6. Galef, B.G., Jr. and Clark, M.M.: Social factors in the poison avoidance and feeding behavior of wild and domesticated rat pups. *Journal of Comparative and Physiological Psychology*, 75:341–347, 1971.
7. Galef, B.G., Jr. and Clark, M.M.: Parent-offspring interactions determine time and place of first ingestion of solid food by wild rat pups. *Psychonomic Science*, 25:15–16, 1971.
8. Galef, B.G., Jr. and Clark, M.M.: Mother's milk and adult presence: Two factors determining initial dietary selection by weanling rats. *Journal of Comparative and Physiological Psychlogy*, 78:220–225, 1972.
9. Galef, B.G., Jr. and Heiber, L.: The role of residual olfactory cues in the determination of feeding site selection and exploration patterns of domestic rats. *Journal of Comparative and Physiological Psychology*, In Press.
10. Galef, B.G., Jr. and Henderson, P.W.: Mother's milk: A determinant of the feeding preferences of weaning rat pups. *Journal of Comparative and Physiological Psychology*, 78:213–219, 1972.
11. Galef, B.G., Jr. and Sherry, D.F.: Mother's milk: A medium for the transmission of cues reflecting the flavor of mother's diet. *Journal of Comparative and Physiological Psychology*, 83:374–378, 1973.
12. Garcia, J. and Koelling, R.A.: A comparison of aversions induced by X-rays, drugs and toxins. *Radiation Research Supplement*, 7:439–450, 1967.
13. Hall, K.R.L.: Observational learning in monkeys and apes. *British Journal of Psychology*, 54:201–226, 1963.
14. Leon, M. and Moltz, H.: Maternal pheromone: Discrimination by preweaning albino rats. *Physiology and Behavior*, 7:265–267, 1971.
15. Leon, M. and Moltz, H.: The development of the pheromonal bond in the albino rat. *Physiology and Behavior*, 8:683–686, 1972.
16. Leon, M. and Moltz, H.: Endocrine control of the maternal phenomone in the postpartum female rat. *Physiology and Behavior*, 10:65–67, 1973.

17. Richter, C.P.: Experimentally produced behavior reactions to food poisoning in wild and dometicated rats. *Annals of the New York Academy of Sciences,* 56:225–239, 1953.
18. Rozin, P.: Specific aversions and neophobia resulting from vitamin deficiency or posioning in half-wild and domestic rats. *Journal of Comparative and Physiological Psychology,* 66:82–88, 1968.
19. Rozin, P.: Adaptive food sampling patterns in vitamin deficient rats. *Journal of Comparative and Physiological Psychology,* 69:126–132, 1969.
20. Rozin, P. and Kalat, J.W.: Specific hungers and poison avoidance as adaptive specializations of learning. *Psychological Review,* 78:459–486, 1971.
21. Rzoska, J.: Bait shyness, a study in rat behavior. *British Journal of Animal Behavior,* 1:128–135, 1953.
22. Steiniger, von F.: Beitrage zur Soziologie und sonstigen Biologie der Wanderratte. *Zeitschrift fur Tierpsychologie,* 7:356–379, 1950.
23. Telle, H.J.: Beitrag zur Kenntnis der Verhaldtensweise von Ratten, vergleichend dargestellt bei, *Rattus norvegicus und Rattus rattus. Zeitschrift fur Angewandte Zoologie,* 53:129–196, 1966.

DISCUSSION

NOWLIS: I just wondered if you ever observed the poisoned rats to defecate around the poisoned food?

GALEF: As you may know Steininger,[22] who some years ago described an instance of traditional poison avoidance in wild *R. norvegicus,* hypothesized that animals which had learned to avoid ingesting a toxic bait warned off naive conspecifics by urinating and defecating in it. In contrast, our laboratory observations indicate that both domestic and wild rats deposit excreta in and around a food source they are utilizing, not one they are avoiding, and that the presence of fresh excreta will attract pups to a feeding site, while old excreta has no observable influence on feeding site selection.[9]

STANLEY: Might there be only one mechanism, not three, and many different ways to bring that mechanism into play? Could the mechanism be eating a food with a distinctive taste, or with a distinctive form?

GALEF: That's an interesting question. My present feeling is that there are two fundamentally different mechanisms involved here. The direct transmission of flavour cues through mother's milk to the pups is very different from the attraction of pups to a location at which food is available. Adults seem to be able to attract pups to a feeding site in two ways, either by being physically present at it or by excreting in its vicinity.

STANLEY: Will the cues work without food ingestion?

GALEF: The pups prefer to explore in a scent marked area or in the vicinity of adults even if no food is present.

STANLEY: Suppose you expose them to the odor or scent of the feces and never let them eat. Would that be enough to lead them to prefer similarly smelling food? If it were, a mechanism based on olfaction alone could be assumed because the common factor, eating, had been excluded.

GALEF: I think I see what you are getting at. Michael Leon and I are currently engaged in an experiment which relates to all three of your questions. Our preliminary data indicate that exposure of isolated preweaning

albino rats to an arbitrary odor, we are using peppermint extract, is sufficient to cause exposed pups to prefer peppermint-extract flavored diet at weaning. If this effect holds up, as I expect it will, it suggests that all of the mechanisms I've been discussing today are in a sense similar, as you suggested in your first question. They all involve the familiarization of the pups with the taste and smell of a food. So one can conceive of the ingestion of flavors in mother's milk and the tendency of pups to approach adults at a feeding site as alternative means by which pups become familiar with a diet and come to prefer it. As you suggested in your second question, ingestion would not be a necessary condition for the development of familiarity with a diet, at least not for the development of familiarity with its smell. In answer to your present question, our earlier data [10] suggest that differences in the smell of feces as a function of diet are not sufficient to familiarize pups exposed to that feces with the diet of the animal excreting it. However, I think more work is needed before I'd feel confident in totally rejecting that notion.

LE MAGNEN: It is a transmission between adults and young. My coworkers (J. Danguir & S. Nicolaidis) have shown that the same transmissions may be observed between adults.

GALEF: We've looked at the ability of adults to influence the dietary preference of conspecifics in situations very similar to those we use with pups. In general, the effects seem much weaker than with pups. Adult rats seem to be less susceptable to social influence in choosing diets.

LE MAGNEN: It is a sort of mother conditioning?

GALEF: Not directly. Rat pups of weaning age preferentially approach an area scent marked by an adult or containing an adult. It doesn't seem to matter if that adult is their mother or not. Of course, experience of the mother during infancy may be a necessary precondition for the emergence of these social tendencies at weaning. We don't know.

JACOBS: Although it is clear that early experience can produce strong effects on food choice when tested at weaning and shortly thereafter, other studies [1,2] have shown the effects to be transient when tested later in life. In your experiments, you have shown post-weaning effects. What happens if you test these animals as adults?

GALEF: Unfortunately, we have never left the adults and pups together for more than 40 days so we really don't know. My suspicion is that if you left a colony of wild rats intact until the pups reached adulthood, you might still see an effect of the first generation's learned feeding site preference on the feeding behaviour of the second generation. Steiniger's field data suggest this influence might last indefinitely.

JACOBS: While the reinforcement is continued?

GALEF: In Steiniger's situation the poison was left in the avoided diets, but he claimed that the second generation never ate any of the poison bait so long as members of the first generation were still alive.

JACOBS: I asked this question be-

---

[1] Warren, R.P., and Pfaffmann, C.: Early experience and taste aversions. *J. comp. physiol. Psychol.* 52:263–266, 1959.

[2] LeMagnen, J.: Advanced studies on the physiological control and regulation of food intake. In Stellar, E. and Sprague, J.M. (Eds.), *Progress in Physiological Psychology*, New York, Academic Press, 1971.

cause social interaction in the colony may itself provide many sources of reward and punishment that could well influence food preferences. A mechanism for this is suggested by the classic studies of Bruce,[1] and Seward,[2,3] who worked on the role that environmental factors can play in the establishment and modification of dominance/submission hierarchies in the rat.

GALEF: One would, of course, expect aggressive interactions at a preferred feeding site to result in the dispersal of subordinates to less desirable feeding locations. However, as Bruce indicated, aggressive competition for food is rarely seen in rats unless the necessity for food competition is imposed on the subjects by the experimental situation. We were careful to provide food sources of sufficient size to allow simultaneous feeding by a number of animals and, as is generally the case in established rat colonies [3] aggression among adults was very rare. Occasionally adult females would "drub" pups (23 days or older) when pups got between them and food.[6] Our impression was that pups punished in this way tended to approach the feeding site when the aggressive individual was absent. However drubbing clearly did not cause the pups to abandon the adult utilized feeding site for an available alternative.

In summary, I wouldn't expect aggressive interactions to disrupt the tendency of successive generations to feed at the same site so long as an adequate supply of food was available there.

[1] Bruce, R.H.: An experimental analysis of social factors affecting the performance of white rats. III Dominance and cooperation motivated by water and food deprivation. *J. comp. Psychol.* 31:395–412.

[2] Seward, J.P.: Aggressive behavior in the rat. II An attempt to establish a dominance hierarchy. *J. comp. Psychol.* 38:213–224, 1945.

[3] Seward, J.P.: Aggressive behavior in the rat. IV Submission as determined by conditioning, extinction and disuse. *J. comp. Psychol.* 38:51–75, 1945.

Chapter 17

TASTE EXPERIENCE AND THE DEVELOPMENT OF A DIETARY PREFERENCE FOR SWEET IN HUMANS: ETHNIC AND CULTURAL VARIATIONS IN EARLY TASTE EXPERIENCE

Norge W. Jerome

The trend toward introducing supplementary foods to infants at increasingly earlier ages has been noted by many authors.[1,11,13,15,21] As early as the first month, supplementary baby foods, primarily juices, are included in the infant's diet.[21,33] Some authors have expressed concern over the sucrose content of these early diets—especially that of commercially prepared strained and junior foods.[1,39]

Analysis of proprietary foods ingested by infants during the early months of life reveal that added sucrose makes up 49 percent of the carbohydrate content of the diet. Fruits, juices and desserts—those items containing added sugar furnish 18–20 percent of the total calories of the 3–9 month old infant's diet.[39]

These developments have led to the postulation that widespread use of highly sweetened foods during the early months of life may predispose the infant to a taste preference for sweet foods in later life.[1,25] However, experimental data reveal that newborn human infants are not only able to preceive sugars but also show a preference for "sweets." [24,30] According to Maller and Desor,[24] "the preference for 'sweets' appears very early in development and is relatively independent of experience." Should one be concerned about developing a dietary preference for sweet among infants if such a preference is already established at birth?

If the preference for "sweets" as shown among human newborns in the Nisbett and Gurwitz and Maller and Desor studies [24,30] is an immutable and universal human trait, lacking additional experimental data, further evidence of such a trait might be obtained from ethnographic cross-cultural data on infant feeding practices. If the preference for "sweets" among human newborns is a universal human trait, infant diets in most cultures, even among hunter-gatherer and other isolated groups will contain items to indulge that preference. Ethnographic materials provide additional advantages. If early experience with dietary sweets is critical to the development of a preference for sweets, then cultures lacking such a tradition will either reject or be neutral to dietary sweets in post-contact situations.

This paper will provide data on in-

fant feeding practices in various cultures. It attempts to integrate the fragmentary material from the literature and so provide a cross-cultural overview of practices showing variations in use of dietary "sweets" in early life. It will also describe some contemporary dietary practices involving sweets for groups whose early taste experience and traditional diet included few "sweets." This type of overview should provide the uninitiated a better understanding of similarities in taste preferences (as expressed by the use and consumption of dietary sweets) among groups with diverse early taste experiences in a variety of cultures.

"SWEETS" IN PRE-LACTEAL AND NEONATAL DIETS

Data on infant and child feeding and rearing patterns reveal that infants in a great many cultures—modern, high income, industrialized and in traditional, low-income, non-industrialized—are given sweet liquid in some form soon after birth. Sugar and honey are the most common items served. This practice has been referred to as "pre-lacteal feeding." The infant continues to receive sweets in his diet throughout the first year of life and presumably thereafter. The objective is to provide a diet that is protective, appropriate, pleasant and palatable as defined by the culture. Other cultures without a tradition of "pre-lacteal" feeding of dietary sweets nurture their infants in accordance with ecological and cultural dictates. Both traditions provide insight on the concept of the development of dietary preference for sweet among humans.

In a great many cultures, prohibitions surrounding the colostrum of the new mother, the meconium of the newborn, and the physiology of lactation and of the newborn have dictated practices which favor the early introduction of "sweets" in the feeding of the newborn. These prohibitions or taboos are present in peasant or transitional societies as well as among the most technologically advanced. They are manifest in what may be termed "neonatal feeding rites" which may last from one day to one week prior to breast, hand, or bottle feeding. The duration of this period is dependent upon culture and the beliefs and traditions on which the practice is based.

The neonate ingests clear or viscous liquids which are distinctively sweet. The composition and viscosity of the liquid are associated with cultural heritage; however, the one constant factor—sweetness—is provided by either honey or sugar. In reference to the practice of giving sugar and water to the newborn in the U.S.A., Wickes [43] linked the custom to a survival of practices which had been initiated by the Greeks and Romans and continued by the English and French in the 17th Century. Even today, one of the first experiences of the newborn in U.S. hospitals is the taste of 5 percent glucose solution. Physicians defend this practice by stating the need to assess the functioning of the newborn's digestive system immediately after birth and by asserting that "the newborn tolerates glucose better than water." Nisbett and Gurwitz [30] and Maller and Desor [24] have provided experimental data in

support of this heretofore subjective observation.

The practice of giving babies sugar, honey, oil and/or spices immediatly after birth has been described for many countries in South and Central America. These neonatal feeding practices are based on beliefs held about both the meconium and colostrum. The Reichel-Dolmatoffs' description of neonatal feeding practices in a Colombian mestizo village [35] is typical.

> . . . If the mother's milk should not have appeared during the first day after birth, the baby is given a few spoonfuls of lukewarm water containing some raw sugar, prepared and administered by the midwife or some other adult woman, but as soon as the milk begins to flow the infant is breast fed . . . Practically all women who have milk nurse their babies, but among the poorer people there are many who are undernourished and cannot breast-feed them. If they can afford it, they will try bottle-feeding with powdered milk, but as this product is very expensive by local standards, they usually prepare sweetened rice water, or simply lukewarm sugar water, sometimes containing a bit of milk. Milk, when available is always diluted with water and heavily sweetened. . . .
>
> At about two weeks after birth, the baby is given little spoonfuls of sweetened rice water, and tea of dried rose leaves or of *siempreviva (Gomphrena* sp.), sweetened with sugar . . . "so he will have a healthy color. . . ." [p. 80]

Huenemann's account[16] of infant feeding practices in a Peruvian village revealed the practice of giving some sort of sugar solution soon after birth until such time as the breast was given. Some infants received plain sweetened water, while others were given sweetened water supplemented with almond oil, anise, cinnamon, molasses, or herbs. Wellin[42] also made observations on the liberal use of sugar in early feeding in another Peruvian village. Behar's report [4] on the dietary pattern of infants in a Mayan Indian community made reference to "liquids—usually water with sugar given to most children during the early weeks after birth."

As recently as one generation ago in my native island Grenada, the newborn was given a mixture of honey, olive oil and spices soon after birth. In addition to its cleansing properties, the highly viscous mixture, locally referred to as "Luck" was believed to bring good luck to those who ingested it.

The Polynesian neonatal feeding tradition varies only slightly from that described for the Caribbean and Latin American countries. Mead [26] provided a bird's eye view of infant feeding in Samoa. In addition to mother's milk, babies were given sugar cane juice and papaya during their first week. Unfortunately, the report does not indicate whether the ingestion of sugar cane juice preceded lactation.

In South and South-east Asia, neonatal feeding practices are largely associated with beliefs about colostrum. Robinson,[36] reporting about India in general, stated that during the first 24 hours after birth, water with sugar or honey is given prior to putting the infant to the breast. Arora and Kual [2] observed similar practices in central Indian communities. Infants were never put to the breast before 24 hours after birth; honey, however, was the most popular prelacteal feed among all communities. Minturn and Hitchcock [27] made similar observations in the study of child rearing in the village of Khalpur. Their ethnographic report also re-

ferred to a dish served the newborn which consisted of about one cup boiling water, caraway seed, two puffed sugar candies and a pinch of ashes. Rao et al [34] confirmed this observation for southern India. Dates and castor oil were added to the list of items given during the first three days after birth before the baby is put to the breast.

The custom in urban West Bengal is essentially similar; Mukherjee [29] reports that during the first few days after birth when milk flow was not sufficient the neonate received plain water, but more commonly sugar or sugar-candy (misri) water.

Burmese and Pakistani practices are essentially similar to those observed in various Indian communities. In Upper Burma, the neonate ingests honey served from muslin dipped in honey.[12] Similarly, Pakistani neonates are fed a mixture of ghee, sugar and honey mixture given with the finger.[17]

The practice of giving sweet liquid to neonates immediately after birth is probably more widespread than the above reports indicate. Without an intimate knowledge of the culture, researchers may fail to uncover such practices. Moreover, most of the research on infant and child feeding practices and patterns were conducted by individuals or teams of workers who were primarily concerned with the nutritional contribution of breast, bottle or supplementary feeding to the infant's health status.

As has been shown for selected Western, Latin American and Asian cultures, the custom of introducing "sweets" into the newborn's diet is linked with cultural views on the physiological, medicinal and sensory needs of newborns. Goals of achieving both biological and sensory needs are well described by Childs [6] in communicating his observations on the Umbundu.

> . . . from the time of its birth a little gourd or pot of beer (ocisangua) is set aside for the baby. It is tasty, very tasty. Into that beer they throw some sweet-smelling medicine. The medicine helps that the stomach may not ache and that worms may not come. The chief purpose of the beer and the sweet-smelling medicines is to tempt the baby to eat and drink, for *it is believed that a baby is born with its senses of taste and smell fully developed* . . . (emphasis mine)

In describing the quantitative aspects of supplementary feeding, Behar [4] commented appropriately by stating "the real intent of mothers was to develop in the children a *taste* for foods other than breast milk and *to accustom them to their use* (emphasis mine), rather than to provide added nutrient."

"SWEETS" IN POST-NEONATAL FEEDING PATTERNS: THE FIRST YEAR

Low income countries

A cursory look at supplementary feeding practices * during the post-neonatal period indicates that those engaged in infant and child feeding sought to achieve four basic goals—palatability, nutrition, health, and perpetuation of adult foodways. The "proper taste" was achieved by including sugar and other flavoring ingredients, while nutrition and health were guaranteed by excluding those

* Practices which exclude breast feeding e.g. feeding by hand, bottle, jar, dish, etc.

items deemed harmful to the infant, usually meat, fish and eggs—bland foods of high protein content which were generally thought to cause worms. Staples of the adult diet were prepared in ways which could be readily accepted and utilized by the infant. Thus, cereals, roots, fruits, and vegetables were mashed, premasticated, blended, creamed and sweetened to conform to the adult's view of consistency and taste appropriate for the human infant. These basic principles are illustrated in descriptions of supplementary diets in a variety of cultures.

Jelliffe [17,19] and Swaminathan's [40] comprehensive reviews of infant feeding in 40 countries in the subtropics and tropics (low income countries) revealed some basic similarities in the supplementary diets designed for infants. Among these are breast feeding, the introduction of semi-solid foods during the second semester, or second year in the majority of Far Eastern countries and in the Pacific Islands; and the widespread and liberal use of sugar in infant food preparation or in products served infants. The maize porridge of West Africa parallels the cornstarch pudding "mehallabiah" of the Middle East countries, the "chipatties" of Pakistan, the cassava and millet gruel of Central Africa, the maize gruel of East Africa, and maize porridge of South Africa. These products are also similar to the cereal and cassava gruels (atoles) of South and Central America, the rice porridge or gruel of India, Ceylon and South-east Asia, the banana gruels of South-east Asia and the cornmeal, arrowroot and oatmeal porridge of the West Indies. The chucula of Panama (a popular thick drink made of maize flour, ground cocoa beans, cane juice or sugar and water) fed infants of the San Blas Indians of Panama as described by Jelliffe et al [18] also fall in this category. In summarizing the supplementary diets in Africa, Davies [9] states that while the diet varies from place to place, the supplements consist only of watery gruels or sweet foods like the banana.

Most of the preparations are well fortified with sugar; in addition, sweetened condensed milk remains a staple in most diets. Milky tea, sweetened with large amounts of sugar is a staple of the Middle East, some North and Central African countries and the Pacific Territories; while sweetened bush tea is a staple of many West Indian islands. So-called bland foods—meat, unsweetened milk and eggs are conspicuously absent.

Is early experience with sweet through supplementary infant preparations peculiar to the tropics, subtropics and low income countries? The data would indicate that while the degree of sweetness for any given item may vary in accordance with cultural food traditions and preferences, the total amount of sugar added to infant supplementary foods may not differ substantially between high and low income countries.

High income countries

In modern societies, breast milk remains one of the very few items in the infant's diet which has resisted the direct addition of sugar. Consequently, if current trends in early diet supplementation continue, opportunities for including sweets in the

infant's diet in early life will increase.

Frequency of breast feeding in the U.S.A. was estimated to be 26 percent in 1965. Since 1958, the frequency of breast-feeding at age one month has remained constant at about 20 percent of the infant population. By age 4 months breast-feeding decreased to 8 or 10 percent of the infant population and by age 6 months only about 5 percent of infants were breast-fed.[13] It is believed that recent developments among U.S. college-educated middle and upper income women and lay organizations to reverse this trend will change these figures. It is doubtful, however, that teenage mothers and those of U.S. ethnic minority groups with very limited income will follow this trend. The decline in infant breast-feeding is typical of the modern industrialized world.

Results obtained from U.S. regional and ethnic studies on infant and child feeding patterns and practices support the data obtained from national studies. A recent report on a study of nutrition of infants and pre-school children in the North Central Region of the U.S. showed that 75 percent of the 2,000 mothers in the study bottle-fed their children exclusively; 22 percent breast-fed their infants, and 3 percent employed a combination of breast- and bottle-feeding from birth. Two-thirds of the mothers who breast-fed their infants discontinued the practice at 3 months.[10] In the U.S., the trend away from breast-feeding and toward formula feeding and the early introduction of supplementary foods transcends ethnic groups. Jerome and co-workers [21] obtained data on infant feeding practices from a predominantly Afro-American community in the north central region. Their findings were very similar to the national and regional figures. By the time the infant is 3–4 weeks old, in addition to a commercial or evaporated milk formula, it has begun to receive orange juice, and cereal (usually rice) with added sugar. At 3 months, infants are fed commercially prepared vegetables, fruits and desserts (the latter two with added sugar).

While commercially prepared infant formulas, other foods marketed for infant consumption, and medications like cough syrup and vitamin drops are prepared with what might be considered unnecessary sugar, United States folk practices of only a few decades ago endorsed the use of large amounts of sugar as an ingredient in baby foods, and as an infant pacifier. The "sugar teat"—a nipple dipped in sugar and handed to the irritable child to suck on in order to pacify him, and the "sugar sack"—a small cloth sack filled with sugar and given to the infant to suck on in order to soothe and comfort him, go back just one generation. The goal was to comfort, please, and reassure the infant.

"SWEETS" IN EARLY CHILDHOOD FEEDING PATTERNS OF HIGH INCOME COUNTRIES

Inclusion of dietary sweets continues through early childhood. Analyses of food energy reported by Eppright et al [10] showed that intake of candy and soft drinks was positively related to the food energy constituents of the diet, particularly carbohydrates. As the children became

older and larger they obtained more food energy from soft drinks and candy. "Mothers," they reported, "used sweet foods as rewards, and deprivation of sweet foods as a punishment. Yet, one of their major concerns was that the children ate too many sweets."

Sweet foods appear to be a dietary staple in many technologically advanced countries. Samuelson's [37] report on health and nutrition of 1,401 children aged 4 to 13 residing in two rural areas and one city in a northern Swedish county revealed a tendency to consume a diet rich in fat and refined sugar products. A significant portion of the food energy was obtained from sugar products.

TRENDS IN SUGAR CONSUMPTION

In discussing patterns and trends in carbohydrate consumption Yudkin [45] demonstrated associations between national wealth and the purchase of more sugar-containing manufactured foods. Per capita consumption of commercially prepared sugar-containing foods increases with income. As a country moves from poverty to affluence, there is a small rise in protein intake, a considerable rise in fat consumption, and a drop in starch consumption that is about the same as the rise in sugar. Much of the sucrose in the diets of the high-consuming countries is found in manufactured foods such as candy, jams, jelly, soft drinks, ice cream, cakes and cookies. For Western countries, the average intake of sugar is usually between 15 and 20 percent of the energy value of the diet.[46] Yudkin fears that in the poorer countries the consumption of sugar and sugar-containing foods may be responsible for malnutrition, especially for protein deficiency in children. In the wealthier countries, there is evidence, he says, that sugar and sugar-containing foods contribute to several diseases, including obesity, dental caries, diabetes mellitus and myocardial infarction.[47] Cohen's studies associated with Yemenite immigrants in Israel support these fears.[7,8]

Information on specific countries show similar trends. Data reported by the U.S. Department of Agriculture [41] showed that poorer families (<$1,000 to $5,000 per year) consumed more household sugar than did higher-income families, who consumed much more of their sugar in the form of commercially prepared foods.

The association between income level and consumption of dietary sweets was shown in Owen's study.[31] Cereal grains were major contributors of iron and calories in children's diets. Within this broad food group, breads, rolls, biscuits, cereals, pastas, and grain mixtures contributed progressively fewer calories as socio-economic level increased while cakes, cookies, pies, and sweet rolls contributed more calories for children in high socioeconomic groups.

Are the national trends in the use of sugars and sugar manufactured goods manifestations of early experiences with sweets? Or do they subtly express economic and technologic states (resource distribution) within and among countries? Some clues might be obtained from additional ethnographic reports of groups whose traditional diets are relatively sugar-free.

SUGAR-FREE NEONATAL AND INFANT DIETS

The local ecology, level of technology, and relative freedom of taboos surrounding birth are closely linked to the composition of neonatal and infant diets. Studies of hunter-gatherers show that the diets of these nomadic groups are based on the local ecology and are relatively free of sugar. Honey and ripe fruit are practically the only items with sugar content served to infants in hunter-gatherer groups.

The Hadza (or Watindiga) are a small hunting and gathering group living in an area adjacent to Lake Eyasi in northern Tanzania (Tanganyika). It is believed that the Hadza may be related to the Bushmen of South Africa. Their diet consists of grain, fruits, seeds and berries. Infants are breast-fed for long periods, usually until another pregnancy. In addition to breast milk the infant is fed rendered soft fat as from the zebra, raw or cooked bone marrow, seeds of the baobab fruit, and honey. Overall, their nutritional status is said to be excellent and dental caries are almost absent.[20] It would be extremely useful to secure information on the Hadza's response to "sweets" in post-contact situtations.

The diet of the Bushmen of the Kalahari desert is also solely dependent on the local ecology and therefore virtually sugar-free. Mongongo nuts and meats form the mainstay of the diet; included are small quantities of roots, melons, edible gums, bulbs and dried fruits.[22] The Samburu, a nomadic Masai-speaking, Nilo-Hamitic tribe of northern Kenya represents but one other group with no known history of dietary sweets.[38] Milk is the staple diet of the males in the community, and meat is the only major item included in the diet. Vegetables play practically no part in the diet and maize-meal ("posho"), while eaten in many settlements, is consumed mainly by married women and young children. Infants are put to the breast soon after birth and thereafter, on demand. Supplementary feeding with milk starts a week later, or even earlier should the mother have little or no milk of her own. At about 18 months meat is introduced, and thereafter milk and meat form the usual diet. LeVine and LeVine [23] have described the details of traditional neonatal feeding practices of the Nyansongo of Kenya. They have no taboos against the colostrum and nurse the newborn at the breast from its first day. Prior to breast feeding, juice cooked from the leaves of a pumpkin squash plant is fed to the infant "to wash out its feces"; later, a few hours after birth a bit of liquid porridge is put into the infant's mouth to stimulate the appetite.

Sucrose is obviously not a necessary ingredient in the diets of these groups. Apparently, the human infant is able to thrive with minimal dietary sucrose. However, how would similar groups with traditional low sucrose diets respond to the introduction of sucrose, bakery goods, candy and other "sweets" in post-contact situations?

TRANSFORMING LOW-SUGAR TRADITIONAL DIETS TO HIGH-SUGAR NON-TRADITIONAL DIETS

Diets of infants and adults change when contact with Caucasians and the "modern" Western world bring new foods to the traditional dietary. Gonzalez [14] has provided an excellent review of the changing dietary patterns of North American Indians. In all areas, many of the items currently available at stores or trading posts and which are purchased for most meals include sugar, molasses, syrup, honey, tea and coffee. Gonzalez asserts that the increasing dependence upon purchased supplies results in the popularity of soft drinks and sweets, such as sweet rolls, cakes, doughnuts and others that are available in every store catering to Indians. A similar case can be made for the Maoris of New Zealand;[32,44] and for the northern Alaskan Eskimos.[5,28]

To date, there have been no reports on any group with a non-sugar tradition rejecting sugar, sweetened condensed milk, sweetened beverages, sweetmeats, pastries, confectionary or other sweet dietary items after their introduction into the culture. In fact, a recent study on sucrose intolerance in northern Alaskan Eskimos revealed that sucrose intolerant individuals continued to consume sucrose despite the discomforts associated with the offending items.[5]

For many cultures, traditional meat-vegetable-fruit diets supplying negligible amounts of sucrose from plant sources are being transformed to diets which are extremely high in sucrose. It can be surmised that for those groups, early taste experience with "sweets" was not a necessary precondition for the development of a preference for "sweets." The physical, social and technologic environments which include marketing and distribution practices seem to be more closely linked to a preference for "sweets" than early stimulation of taste buds.

SUMMARY AND DISCUSSION

It is obvious that infant diets are as varied as the ecology and cultures from which they arise. Many cultures provide pre-lacteal, neonatal feeds for the newborn; many of these feeds have a sugar and honey base, others do not.

Despite basic cultural differences in infant feeding, first-year diets of diverse groups are basically similar in that they consist of items from the adult dietary which had been modified slightly to aid digestion and gain acceptance by infants. In many cultures the latter goal is achieved through sugar. Do these practices aid in developing a dietary preference for "sweet"? The answer is not clear since populations with different dietary histories also demonstrate a preference for sweet. Apparently, if "sweets" are available groups select and use them, to a greater or less extent, in a variety of home-prepared or manufactured foods, depending upon the resources available to them.

Would knowledge of an almost universal preference for dietary "sweets," coupled with the health hazards associated with high sucrose consump-

tion aid in redirecting product development, distribution patterns, marketing strategies and consumption practices? Any change in consumption practices to achieve desired health goals would require responsible action by provider groups (government, industry and other change agents) and consuming populations.

Change in food consumption styles may be readily achieved through infant diets. Observations of immigrants and in-migrant groups have indicated that in their effort to provide the best for their infants, mothers readily adopt the infant feeding styles of those they emulate. Aykroyd and Hossain's [3] description of the artificial feeding regimen adopted by new Pakistani immigrant mothers in Britain furnishes additional data on this subject. The diets of the infants born to recently migrated Pakistani mothers were changed to conform with current British methods although the Pakistani adult diet still consisted almost entirely of traditional foods. My ethnographic data on infant feeding of Afro-American infants in Kansas City support these findings. If mothers are convinced that a proposed diet is suitable, safe, pleasant, palatable, convenient and stylish they readily adopt it for their infants.

REFERENCES

1. Anderson, T.A. and Fomon, S.J.: Commercially prepared strained and junior foods for infants. *J. Am. Diet. Assoc.,* 58: 520–527, 1971.
2. Anova, D.D. and Kual, K.K.: Feeding practices during the first five years among central Indian communities. *Ind. J. Pediat.,* 40: 203, 1973.
3. Aykroyd, W.R. and Hossain, M.A.: Diet and state of nutrition of Pakistani infants in Bradford, Yorkshire. *Br. Med. J.,* 1:42, 1967.
4. Behar, M.: Prevalence of malnutrition among preschool children of developing countries. In Scrimshaw, N.S. and Gordon, G.E. (Eds.), *Malnutrition Learning and Behavior,* Cambridge, MIT Press, 1968.
5. Bell, R.R., Draper, H.H. and Bergman, J.G.: Sucrose, lactose and glucose tolerance in northern Alaskan Eskimos. *Am. J. Clin. Nutr.,* 26:1185, 1973.
6. Childs, G.M.: *Umbundu Kinship and Character,* London, Oxford U. Press, 1949.
7. Cohen, A.M., Bavly, S. and Pozmanski, R.: Change of diet of Yemenite Jews in relation to diabetes and ischemic heart disease. *Lancet,* 11: 1391, 1961.
8. Cohen, A.M.: Lessions from diabetes in Yemenites. *Isr. J. Med. Sci.,* 7: 1554, 1971.
9. Davies, J.N.P.: Nutrition and nutritional disease. *Ann. Rev. Med.,* 3: 99, 1962.
10. Eppright, E.S., Fox, H.M., Fryer, B.A., Lamkin, G.H., Vivian, V.M., and Fuller, E.S.: Nutrition of infants and preschool children in the north central region of the U.S.A. *World Rev. Nutr. Diet.,* 14: 269, 1972.
11. Filer, L.J., andM artinez, J.A.: Intake of selected nutrients by infants in the United States. An evaluation of 4,000 representative six month olds. *Clin. Pediatr.,* 3: 633–645, 1964.
12. Foll, C.V.: The perils of childhood in upper Burma. *J. Trop. Pediat.,* 4: 122, 1958.
13. Fomon, S.J.: *Infant Nutrition,* Philadelphia, W.B. Saunders Company, 1967.
14. Gonzalez, N.L.: Changing dietary patterns of North American Indians. In Moore, W.M., Silverberg, M.M. and Read, M.S. (Eds.), *Nutrition, Growth and Development of North American Indian Children,* DHEW Pub. No. (NIH) 72: 26, Supt. Documents, U.S. Govt. Printing Office.

15. Harris, L.E. and Chan, J.C.M.: Infant feeding practices. *Amer. J. Dis. Child.,* 117:483–492, 1969.
16. Huenemann, R.L. and Collazos, C.: Nutrition and care of young children in Peru. *J. Am. Diet. Assoc.* 30:559, 1954.
17. Jelliffe, D.B.: Infant nutrition in the subtropics and tropics. *World Health Organization Monograph,* No. 29, 1955.
18. Jelliffe, D.B., Jelliffe, E.F.P., Garcia, L., and de Barrios, G.: The children of the San Blas Indians of Panama. *Pediatrics,* 59: 271, 1961.
19. Jelliffe, D.B.: Culture, social change and infant feeding: current trends in tropical regions. *Am. J. Clin. Nutr.,* 10: 19, 1962.
20. Jelliffe, D.B., Woodburn, J., Bennett, F.J. and Jelliffe, E.F.P.: The children of the Hadza hunters. *J. Peds.,* 60: 907, 1962.
21. Jerome, N.W., Kiser, B.B. and West, E.A.: Infant and child feeding practices in an urban community in the North-central region. In Fomon, S.J. and Anderson, T.A. (Eds.), *Practices of low-income families in feeding infants and small children with particular attention to cultural subgroups,* U.S. Dept. HEW, DHEW Pub. No. (HSM) 72: 5605, 1972, Supt. Documents, U.S. Govt. Printing Office.
22. Lee, R.B.: Kung Bushman subsistence: an input-output analysis. In Vayda, A.P. (Ed.), *Environment and Cultural Behavior,* New York, The Natural History Press, 1969.
23. LeVine, R.A. and Levin, B.B.: Uyaansongo: a Gussi community in Kenya. In Whiting, B.B. (Ed.), *Six Cultures: Studies in Child Rearing,* New York, John Wiley and Sons, 1963.
24. Maller, O. and Desor, J.A.: Effect of taste on ingestion by human newborns. In Bosma, J.F. (Ed.), *Fourth Symposium on Oral Sensation and Perception,* Washington, Superintendent of Documents, U.S. Government Printing Office, 1973, pp. 279–291.
25. Maslansky, E., Cowell, C., Carol, R., Berman, S.N. and Grossi, M.: Survey of infant feeding practices. *Am. J. Public Health,* 64: 780–785, 1974.
26. Mead, M.: *Coming of Age in Samoa,* New York, Dell Publishing Company, 1928.
27. Minturn, L. and Hitchcock, J.T.: The Tajputs of Khalapur, India. In Whiting, B.B. (Ed.), *Six Cultures: Studies of Child Rearing,* New York, John Wiley and Sons, 1963.
28. Mouratoff, G.J., Carroll, N.V., and Scott, E.M.: Diabetes mellitus in Eskimos. *J. Am. Med. Assoc.,* 199: 961, 1967.
29. Mukherjee, P.S.: Feeding of children in urban west Bengal. *Ind. J. Pediat.,* 26: 467, 1959.
30. Nisbett, R. and Gurwitz, S.: Weight, sex and the eating behavior of human newborns. *J. Comp. Physiol. Psychol.,* 73: 245–253, 1970.
31. Owen, G.: Personal Communication, 1973.
32. Prior, I.A.M.: A health survey in a rural Maori community, with particular emphasis on the cardiovascular, nutritional and metabolic findings. *N.Z. Med. J.,* 61: 333–348, 1962.
33. Purvis, G.: What nutrients do our infants really get? *Nutrition Today,* 8: 28–34, 1973.
34. Rao, K.S., Swaminathan, M.C., Swarup, S., and Patwardhan, V.N.: Protein malnutrition in south India. *Bull. World Health Org.,* 20: 603, 1959.
35. Reichel-Dolmatoff, G. and Reichel-Dolmatoff, A.: *The People of Aritama,* Chicago, The University of Chicago Press, 1961.
36. Robinson, P.: Infant feeding in India. *Quart. Rev. Peds.,* 11: 196, 1956.
37. Samuelson, G.: An epidemiological study of child health and nutrition in a northern Swedish county. *Acta. Paed. Scand.,* Supp. 214, 1971.
38. Shaper, A.G. and Spencer, P.: Physical activity and dietary patterns in the Samburu of northern Kenya, *Trop. and Geog. Med.,* 13: 273, 1961.
39. Stewart, R.A. and Pruvis, G.A.: Intake of nutrients by U.S. infants. Personal Communication, Sept., 1974.
40. Swaminathan, M.: The nutrition and feeding of infants and preschool children in the developing countries. *World Review of Nutrition and Dietetics,* 9:85, 1968.

41. U.S. Department of Agriculture: Econ. Res. Serv., National Food Situation, NFS 128, Washington, Superintendent of Documents, U.S. Government Printing Office, 1968.
42. Wellin, E.: Maternal and infant feeding practices in a Peruvian village. *J. Am. Diet. Assoc.*, 31: 889, 1955.
43. Wickes, I.G.: A History of Infant Feeding Part I, primitive peoples: ancient worlds: Renaissance writers. *Arch. Dis. Child.*, 28: 151, 1953.
44. Wright-St. Clair, R.E. : Diet of the Maoris of New Zealand. *Ecology Food and Nutr.*, 1: 213, 1972.
45. Yudkin, J.: Patterns and trends in carbohydrate comsumption and their relation to disease. *Proc. Nutr. Soc.*, 23: 149, 1964.
46. Yudkin, J., Edelman, J. and Hough, L.: *Sugar*, New York, Peter H. Wyden, Inc., 1972.
47. Yudkin, J.: Sugar and disease. *Nature* 239: (#5369) 197, 1972.

DISCUSSION

Nowlis: Up until, I believe, a century ago there was a practice in Europe of feeding infants pre-masticated foods. The pre-masticated food was transferred from the mother's mouth to the infant's mouth. That fascinated me. I wondered about what sorts of things the adults would choose to pass to the child in this manner. I even wondered whether there was any predigestion of starches to make it more sweet.

Jerome: Yes. Most foods—starches and meats—were premasticated and then transferred from the mother's mouth, either directly or by hand, to the infant's mouth in most societies including European societies. Ptyalin, a starch enzyme present in saliva, acts in breaking down complex cooked starches at that stage to dextrins and maltose (malt sugar).

Nowlis: Is there any culture nowadays that still practices this?

Jerome: Oh yes, of course. This practice still continues among groups whose technology provides no other means for softening foods for infants. The mouth's apparatus—the original blender—is still used among many hunter-gatherer groups, for example.

Nowlis: Are there any indications of how the choice is made? Which foods, for example, are excluded from the infant's diet?

Jerome: Food selection varies from culture to culture. Every culture has a definition of what is appropriate for infants immediately after birth and during the first weeks of life. Cultural definition of "appropriate foods" continues until the child is weaned to adult foods. Often, specific dietary items are considered taboo foods for infants and very young children and, therefore, are excluded from their diets. Meats, fish and eggs, for example, which are said to give children worms are often excluded from the diet of infants and young children who are being raised traditionally in certain African cultures. Those items which are defined as appropriate for the infant and young child and which require pre-mastication or blending are subjected to those procedures. You will note that despite the wide range of baby foods produced commercially in our culture we have not yet seen jars of caviar, oysters, fish, lobster or shrimp. Obviously, in the U.S. these food items are not considered appropriate for babies.

Moskowitz: This possible acculturation of sweet preference is fascinating, and it might be wise to look

for acculturation of other kinds of tastes. Now, much previous data lead you to believe that infants reject, or at least act as if they reject sour. As you know, in our culture sour is not necessarily a pleasing taste in an aqueous solution alone. However, just recently, Sharma and Harry Jacobs and myself found a group of Indian laborers, the Karnatka who have been chewing tamarind fruit, an exceptionally sour tasting material, all of their lives. It was expected that these people who have experience with sour for their entire lives by chewing this fruit as a confection, would have different affective, or different taste responses to it. We tested them on various concentrations of sweet, salt, sour and bitter, both for taste intensity and for taste pleasantness; and lo and behold, they have a normal sensory system, their tongues are the same as ours. However, their affective responses to sour showed that they prefer aqueous solutions of citric acid to an increasing degree, with increasing concentration. They respond like Occidental populations to sweets, as we do to salts and a little bit "strangely" to bitter (they like it at low levels, but dislike it at high levels) but they actually completely enjoy the sour taste of citric acid. It's not a confection, it's simply sour water. There is some kind of acclimitization functioning here. They really like it.

JEROME: I agree. I enjoy tamarind very much although my American friends reject the fruit and find that level of tartness objectionable. I believe that my experience with tamarind from early childhood has made a real difference in my accepting and enjoying that very sour fruit.

I would like to make one further comment on acceptance and rejection of foods and substances by different groups. There are many cultures where weaning is initiated by rubbing the lactating woman's nipples with aloe (a bitter substance) or some other bitter substance. I believe that this practice is very instructive. Women know by experience that the young child will reject the breast after having experienced that unpleasant taste. Once this occurs, the weaning process begins.

We have just discussed various practices of using sugar and other "sweets" to entice the infant to eat. In a great many cultures, then, sweets are used to entice and bitter to repel.

COLLIER: I'll go back to an earlier question. One of the problems of having an infant on a low protein diet involves the food intake. Very often the mothers in these cultures keep adding sugar or anything sweet to the foods in order to increase the child's food intake. This practice, of course, exaggerates the condition.

JEROME: Yes. That indeed is a problem. By enticing the child to eat with added sugar, one lowers the protein quality of the diet.

JACOBS: Whether one is an industrialist analyzing the growth of international sugar markets, or a nutritionist or dentist decrying it, the introduction of refined sugar in the 18th century is considered a critical event in the establishment of a general "sweet-tooth." Although this makes sense in economic terms, you have suggested that cultures exist that are far removed from refined sugar markets by time and/or geography, and yet still demonstrate a "sweet-tooth." Is this true?

JEROME: Quite true. Sweets are im-

portant in the diets of most peoples despite their proximity to or remoteness from refined sugar markets. However, one ought to make a clear distinction between refined and unrefined sugars. Unrefined sugar contains some micronutrients and serves as a vehicle for providing some needed nutrients. This does not hold for the empty calories of refined sugar. Given a choice, unrefined sugar should be selected over refined sugar.

GRINKER: The use of sweeteners or sugar and the incidence of obesity seem to show opposite trends. In both New York City and London, Stunkard [1,2,3] has demonstrated strong inverse relationships between obesity and socioeconomic status. Obesity was six times more prevalent among women of lower than among women of upper socioeconomic status. Obesity was nine times as prevalent by age six in lower than upper class girls. Similar, but less striking differences appear in men and boys. This is a complicated phenomenon, because differences in ethnic background as well as differences in economic and educational background are involved.

JEROME: I have referred to studies which show that high sugar consumption is associated with low or limited income within and among countries (Refer to page 241). A case might be made for obesity in women and girls of low income but whose consumption of sucrose is high. This point needs further investigation.

RODIN: The point was that while the high incidence of obesity in the lower socioeconomic groups is correlated with food habits, it is primarily the ingestion of starches rather than sugars that is unusually high.

JEROME: Data reported by the U.S. Department of Agriculture contradicts this statement. Poorer families (< $1000 to $5000 per year) consumed more household sugar than did higher income families who consumed much more of their sugar in the form of commercially prepared foods—presumably starches.

MISTRETTA: Have you studied baby foods that are commercially available among developed countries, and compared contents at all?

JEROME: No, I haven't, but I will. The new world that preparing for this conference has opened up to me will lead me to numerous new studies of infant feeding practices in low and high income countries.

PFAFFMANN: Since we're discussing various aspects of this matter, there is the interesting class of taste modifiers from west Africa, that occurs naturally in plants. One of them is called miracle fruit, and in fact is used by the population of the area to improve and reduce the sourness of palm wine and sour dough bread. Thus whatever may be the dietary characteristics there, the adults nonetheless will use this material which has only the effect of enhancing sweet flavor.

JEROME: The west African "miracle fruit" could solve some of the problems we face by enhancing sweet flavor without providing food energy or encouraging oral bacteria.

[1] Goldblatt, P.B., Moore, M.E., and Stunkard, A.J.: Social factors in obesity. *JAMA*, 192:1039–1044, 1965.

[2] Silverstone, J.T., Gordon, R.P., and Stunkard, A.J.: Social factors in obesity in London. *Practitioner*, 202: 682–688, 1969.

[3] Stunkard, A.J., d'Aquili, E., Fox, S., and Filion, R.D.L.: Influence of social class on obesity and thinness in children. *JAMA*, 221: 579–584, 1972.

Chapter 18

DIETARY SWEET: EXPOSURE AND PREFERENCE AMONG JAPANESE CHILDREN AND IN LABORATORY RATS

Shisuko Muto

In times of severe food shortage those concerned with maternal and child nutrition can make their professional contribution through seeking substitute foods that satisfy the nutritional requirements of mothers and infants. Fish meal,[23] dried pupea,[17] or soybeans [33,34] can serve as a source of protein and extract of persimmon leaves as a source of vitamin C [18] wild weeds can be eaten in place of traditional vegetables. It is my observation that under such conditions children may grow remarkably well and even be essentially free of feeding problems in the common meaning of the term. In more normal times, when essential nutrients can be selected from a variety of conventional sources, developmental nutritionists concern themselves with the food choice behavior that this opportunity to select implies. Problems arising in connection with food choices include reduced appetite,[8,26,27,28,29,30] obesity, and distortions of food likes and dislikes. [16,41]

The factors controlling food intake are the subject of a diverse literature.[1,3,4,6,7–11,13–15,36,42] Still, it is far from clear to what extent and with what priority the various factors determine what a child will eat. Sophisticated opinions concerning the feeding of children have been presented by authorities on pediatrics [40] and nutrition.[12] Yet, the opinions may not agree or may even be contradictory. Sometimes it is difficult to find studies that support the advice given to mothers.[8] Even when I find what appears to be support for an opinion,[2,5,37,38,39] I am concerned that the conditions of the investigation may be quite different from the circumstances of child rearing as I see them in present-day Japan.

I have approached the subject of food choice behavior and its development through observations of children by either direct or indirect methods and through experimental studies of immature animals whose history of experience with various foods can be controlled.

STUDIES OF CHILDREN

Diet surveys

In 1954–1955, physical examinations, including a tabulation of dental caries, were performed on each of 398 day nursery children from three different regions of Japan, Iwate, Tottori and Wakayama prefectures.

Some of the children from each region, lived on farms and others in fishing villages.[21,22]

The data on dental caries are separately tabulated for each fishing village and farming community in the sample (See Table 18–I). There are no consistent differences due to region or to the type of village. However, children from the fishing village of Wakayama, Fi Wa, are remarkable for their high rate of caries. In this village only 9% of the children do not have caries. In the other villages more than twice this percentage, 18–31%, are caries free. The poor dental health of the children of Fi Wa is further reflected by the high percentage of children with more than 6 carious teeth.[21]

In contrast, one of the other fishing villages, Fi To, shows the lowest percentage of individuals with more than 6 carious teeth. Since the frequency of caries differs so markedly for children from the various fishing villages, the simple notion that the way of life in the fishing village is the cause of the poor dental health cannot be accepted.

Differences in dental health between specific groups, such as those observed here, could arise from differences between the groups with respect to diet. To explore this possibility, trained nutritionists carefully weighed all food consumed during two days by ten randomly chosen 6 year old children in each village. There are differences in the overall quality of the diet (See Table 18–II). In Fi Wa, the intake of such nutrients as calcium, Vitamin A, riboflavin and ascorbic acid is exceptionally low. The children in Fi Wa consume the smallest amount of vegetables and fruits. In addition to the differences in overall quality of the diet there are differences with respect to sweets (See Table 18–III). Sweets including manju (a bread texture confectionary with sweet bean paste inside) are eaten with greater frequency in Fi Wa than in any other village. The consumption of sweets in Fi Wa contrasts sharply with that in the communities with a low incidence of caries, the farm communities in Iwate and Tottori and the fishing village in Tottori.

TABLE 18–I

PERCENTAGE OCCURRENCE OF DENTAL CARIES AMONG 3 TO 6 YEAR OLDS FROM FARMING AND FISHING VILLAGES IN THREE DISTRICTS

Group	*n*	*Number of Caries*		
		0	*1–5*	*over 6*
Iwate				
Farming	43	31	52	17
Fishing	48	25	60	15
Tottori				
Farming	62	29	60	11
Fishing	78	27	68	5
Wakayama				
Farming	62	18	49	33
Fishing	105	9	37	54
Total				
Farming	167	26	54	20
Fishing	231	20	55	25

TABLE 18–II

AVERAGE DAILY NUTRIENT INTAKE OF 10 SIX YEAR OLDS FROM EACH VILLAGE DURING 2 DAYS OF OBSERVATION

	DISTRICT					
	Iwate		*Tottori*		*Wakayama*	
	PRIMARY OCCUPATION					
	Farming	*Fishing*	*Farming*	*Fishing*	*Farming*	*Fishing*
Calories Cal/KG	99	86	87	82	105	108
Protein gm/KG	3.5	3.9	2.7	3.1	3.5	3.7
Carbohydrate gm/KG	19.5	20.2	17.4	19.5	19.6	21.3
Calcium mg	703	449	335	339	556	235
Vitamin A I.U.	2250	2483	1017	1233	2133	720
Thiamine mg	0.56	0.56	0.61	0.41	0.54	0.53
Riboflavin mg	0.75	0.48	0.38	0.40	0.58	0.29
Ascorbic acid mg	42	49	39	30	30	18

TABLE 18–III

THE SNACKS EATEN BY 10 SIX YEAR OLDS FROM EACH VILLAGE DURING 2 DAYS OF OBSERVATION

District and Type of Village	*Sweets**	*Fruits*	*Potatoes & Chestnut*	*Milk*	*Other*	*Total*
Iwate						
Farming	10	14	11	1	17	53
Fishing	17	15	11	4	13	60
Tottori						
Farming	12	6	11	0	12	41
Fishing	15	19	10	2	13	59
Wakayama						
Farming	21	14	1	1	23	60
Fishing	33	6	2	0	16	57
Average						
Farming	14.3	11.3	7.7	.7	17.3	51.3
Fishing	21.7	13.3	7.7	2.0	14.0	58.7

*including Manju, a bun-like confection

The ten children studied in Fi Wa ate sweets between meals on 33 separate occasions. This rate is more than twice that recorded in the three districts with low caries incidence. The eating of fruits shows a complementary pattern with frequencies of 6 in Fi Wa and 19 in Fi To (See Table 18–III).

It is not at all clear which aspect of the striking differences in eating pattern between low and high caries groups accounts for the caries difference. The nutrient intake in Fi Wa is very poor and the consumption of sweets high. I chose to pursue the matter of sweets and confectionary.

Snack buying

To check on the information derived from the intensive study of a few children for a very limited period of time, a study employing an entirely different method was undertaken. In this 1957 study I made a survey of the child's favorite place, the cheap sweet shops, and observed the buying behavior of its customers.[24]

Ten cheap sweet shops (dagashiya) in uptown and ten in downtown Tokyo were selected for study. The visits of children to these shops between the hours of 10 A.M. and 4 P.M. were tabulated. Observations at each shop were made on a Saturday, a Sunday and two week days. In all 431 children visited uptown shops and 669 downtown shops. Children between the ages of five and eight were the most frequent visitors. However, children as old as twelve or as young as one year were observed (See Figure 18–1). Fourteen per cent of the uptown customers and eighteen per cent downtown customers came to the shop twice or more times a day, six times being the maximum.

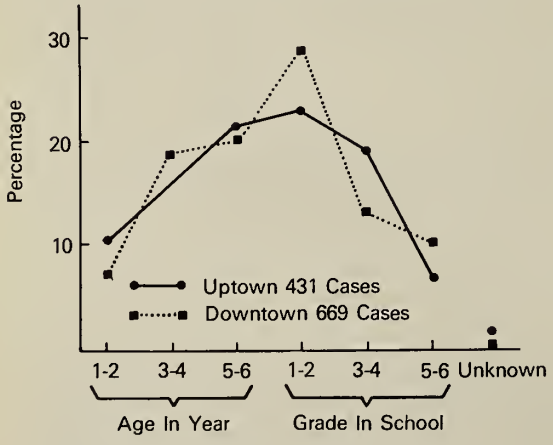


Figure 18–1. The ages of visitors to ten uptown and ten downtown sweet shops. Observations at each shop were made between 10 A.M. and 4 P.M. on two week days, one Saturday and a Sunday. Redrawn from Ref. 24.

In the uptown shops 56.2% of the children bought simple sweets and an additional 29.8% bought sweets which had some special attraction like being formed into an imitation cigar or including a lottery ticket. Only 14% bought only non-food items. A similar pattern was found for the downtown shops as can be seen in Table 18–IV. In both cases more than 85% of the child customers went to the shops for cheap sweets, though some of them seemed to be attracted, to some extent, by small premiums attached to sweets.

Children in institutions

A different kind of information on the topic of snacks and selection of foods is available from questionnaires.[25] We asked 176 children who were under institutional care in Tokyo to give their feelings on snacks. This survey was made in 1960 when an extra five yen per day per child was allowed for snacks. Some foods which were purchasable with five yen in those days were 60 ml milk, 30 to 110 gm fruits, 20 to 50 gm confectionary, etc.

Overall, the greatest percentage of children indicated that sweets are their most favored snack. The percentge of children reporting that sweets are their most preferred snack drops from a high of 53% among preschool children to 30% among senior high school students. At the same time the preference for fruits increases from 19 percent in preschool children to 32 percent in high school pupils.

Of the sweets, chocolate is the most frequent favorite for all school children regardless of age or sex. (See Table 18–V) However, among preschool children, in this sample, the top rated sweets are biscuits followed by gum for boys and crackers followed by gum for girls. (See Table 18–VI) Perhaps chocolate fails to appear as the favorite of these preschool children because their opportunity to become familiar with chocolate was restricted compared that of school age children or pre-schoolers living at home. This speculation is supported by a previous survey [35] in which chocolate was found to be the most preferred snack of 2–5 year old children living with their families. (See Table 18–VII)

TABLE 18–IV

PURCHASES IN UP AND DOWNTOWN SWEET SHOPS

Item	*Uptown %*	*Downtown %*
Sweets alone	56.2	66.8
Sweets with premium	27.7	14.5
Novelty Sweets	2.1	6.3
Toy only	14.0	12.2

TABLE 18–V

SNACK FAVORED BY CHILDREN OF DIFFERENT AGES LIVING IN INSTITUTIONS

		Percentage Favoring			
Group	*n*	*Sweets*	*Bun-like sweets*	*Fruits*	*Homemade snacks*
Preschool	696	53	10	19	18
Elementary	735	45	10	21	24
Junior high	701	31	12	27	30
Senior high	296	30	11	32	27

TABLE 18–VI

TWO MOST FAVORED SWEETS FOR INSTITUTIONALIZED BOYS AND GIRLS OF DIFFERENT AGES

	Preschool	*Elementary*	*Junior high*	*Senior high*
Boys				
1st choice	Biscuit	Chocolate	Chocolate	Chocolate
2nd choice	Gum	Cream sandwich	Cream sandwich	Biscuit
Girls				
1st choice	Crackers	Chocolate	Chocolate	Chocolate
2nd choice	Gum	Gum	...	Gum

TABLE 18–VII

SNACKS MOST WANTED BY 1 TO 4 YEAR OLDS LIVING IN APARTMENTS AND SINGLE FAMILY HOMES

	Apartments	*Houses*
Chocolate	45.0	43.3
Gum	38.5	38.4
Candies	19.2	30.1
Crackers	9.0	18.4
Fruit	10.3	10.0

Note: entries are percentages of 108 for apartment dwellers and 68 for children in single family houses.

Mothers' concerns

Reports by mothers of feeding problems comprise yet another kind of information, concerning children's selective eating of foods.

In one Tokyo survey,[20] 778 middle class mothers with children between two and five years of age were asked how they had treated 26 "problem foods" e.g. sweets, gum, hot spices like pepper or curry, stimulating drinks like coffee, tea, or alcoholic beverages, etc. They also noted their children's health and appetite and reported any cavities.

More than half of the children have an extreme liking for sweets and about one-third of the total group are given sweets frequently. Nearly two-thirds of the children consume sweets in larger amounts than other members of their family. These and other data from this study are shown in Table 18–VIII.

Even though the data come from mother's reports they reflect the relation of the frequency of eating caramels, hard candy and gum to caries. (See Figure 18–2)

Depending on the kinds of sweets, one-fifth to one-third of the mothers report that they started to give sweets to their children before they were one year old and another one fifth to one fourth of the mothers did not remember when they started. (Table 18–VIII)

An indication of cultural similarities and differences with respect to early taste experience can be gained from a survey in which the mothers of American and Japanese preschoolers living in Tokyo report the ages at which they introduced various foods.[32] Some of the data are given in Table 18–IX and X.

ANIMAL STUDIES

Dietary surveys and direct observations of children's food choice behavior raise questions which we now approach through studies of animal behavior. In these studies the dietary intake of developing rats is manipulated by altering their access to various foods and observing the effects on eating patterns and physical growth.

TABLE 18–VIII

HISTORY OF EXPOSURE TO "PROBLEM FOOD" AND EXTREME PREFERENCE AMONG 778 CHILDREN 2 TO 5 YEARS OF AGE

	Chocolate	*Candies*	*Drops*	*Cake*	*Bun*	*Chewing Gum*	*Coca Cola*	*Tea*	*Coffee*	*Pepper*	*Currie*
Extreme Preference	74.6	54.9	48.7	58.0	22.6	75.4	28.9	24.4	35.9	2.2	44.9
Extreme Aversion	0.7	1.9	2.5	2.4	11.1	0.4	15.3	2.4	3.5	32.8	4.7
First offered											
before 1 year	14.8	25.3	19.6	28.2	6.9	1.5	3.7	56.7	1.0	2.2	2.5
do not remember	21.6	20.5	26.6	22.9	35.6	26.7	46.2	20.8	44.6	56.5	39.1
Now offered											
Frequently	47.1	38.8	29.6	31.3	7.6	60.4	4.9	48.2	5.3	1.5	21.0
Sometimes	48.2	50.9	59.5	62.2	58.7	29.5	19.4	39.1	29.3	16.9	55.9
Seldom or never	4.7	10.3	10.9	6.5	33.7	10.1	75.7	12.7	65.4	81.6	23.1
Eaten relative to other family members											
More than	69	67	62	17	8	68	8	2	3	1	2
Same as	24	19	23	67	56	18	42	51	28	36	46
Less than	3	9	10	14	30	6	17	45	63	55	50
Family does not eat	3.	5.	6	2	6	7	34	2	7	9	2

Note: Another 15 items of food are omitted from this tabulation.

TABLE 18–IX

CUMULATIVE PERCENTAGE OF JAPANESE AND AMERICAN CHILDREN HAVING RECEIVED SWEETS, TEA OR SPICES BY DIFFERENT AGES

	Sweets		*Black tea*		*Green tea*		*Coffee*		*Spice*		*Mustard*		*Currie*	
Age	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*
Under one year	54	34	43	15	85	4	9	3	2	22	0	26	4	6
3 years	100	100	91	27	87	8	31	12	14	34	0	46	53	12
Over 3 years	100	100	92	67	93	27	44	18	21	62	1	77	77	50
Not yet introduced	0	0	8	33	7	73	56	82	79	38	99	23	23	50

TABLE 18–X

CUMULATIVE PERCENTAGE OF JAPANESE AND AMERICAN CHILDREN HAVING RECEIVED VARIOUS SOLID FOODS BY DIFFERENT AGES

	Fruit juice		*Soup*		*Vegetables*		*Egg yolk*		*Meat*		*Fish*		*Cereal*	
Age	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*	*Japanese*	*American*
1 month	0	21	0	10	0	11	0	13	0	0	0	0	0	20
3 months	72	65	50	58	8	61	8	63	0	46	3	25	8	83
6 months	100	97	98	84	42	91	82	93	26	73	41	54	75	93
9 months	100	100	100	90	90	99	100	96	75	89	90	79	96	98
12 months	100	100	100	100	100	100	100	100	97	100	100	92	100	98
Over 1 year	100	100	100	100	100	100	100	100	100	100	100	100	100	100
Mean age months	3.3	3.2	4.1	4.4	6.1	3.8	6.1	3.6	8.6	5.3	7.4	5.9	6.2	2.8
Standard deviation	1.1	2.1	1.7	3.3	1.9	2.3	2.1	2.5	2.3	3.1	1.8	3.5	1.8	2.2

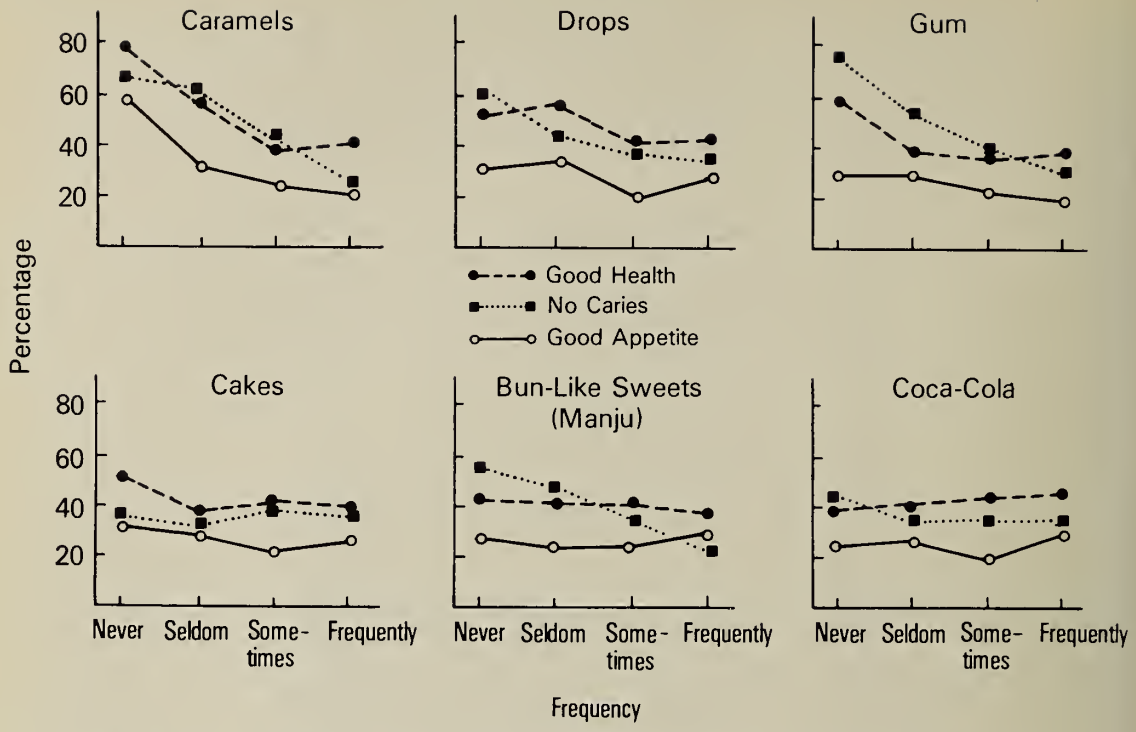


Figure 18–2. Percentage of middle class mothers of 2 to 5 year olds in Tokyo reporting that their children were in good health, had no caries and displayed good appetite, as a function of the frequency with which they ate various snack foods. Redrawn from Ref. 20.

Early versus late introduction of sugar

In a study, previously reported in English,[31] we observed that young rats provided with access to a well balanced diet nonetheless consumed a lot of sugar, reducing their intake of diet but maintaining normal health and growth. When a low protein diet was offered along with the sugar solution, the intake of diet was again reduced but the animal's health and growth suffered. Two questions arise: First, can an adequate protein intake be maintained if young animals are offered both a high and low protein diet along with the sugar solution? Second, does the age at which an animal is first introduced to sugar affect his preferential eating of it?

Newly weaned rats (three weeks old) are divided into three groups all of which are offered both high and low protein diets for six weeks. The high protein diet contains 18 per cent casein, the low protein diet, nine per cent. (See Table 18–XI)

One group is provided with a sugar solution (40% sucrose solution) from the first to the third week. A second group had access to the sugar solution from the fourth to the sixth week. The third, or control group, does not have sugar solution at any time during the experimental period.

The young rats can discriminate between the high and low protein diets,

TABLE 18–XI

PERCENTAGE COMPOSITION OF HIGH AND LOW PROTEIN DIET

	High	*Low*
Corn starch	72	81
Casein	18	9
Soybean oil	5	5
Salt mixture	4	4
Vitamin mixture	1	1
Choline chloride	0.15	0.15

since they always eat more of the high protein diet than the low protein one. However, the ratio between the amount of high and low protein diet consumed during the period of sugar supply is not noticably different from that observed when sugar is not available (See Figure 18–3). Thus, although young rats can discriminate the diets they do not compensate for the drop in protein intake during periods of sugar consumption by preferentially eating the higher protein diet.

Table 18–XII allows for the comparisons among the conditions of this study as well as the comparisons of more immediate interest that can be made with the previous study.[31] Note that during weeks 1 through 3 the ratio of sugar and diet for those animals in the previous study that had high protein diet with sugar since weaning is very close to that of the early exposure animals of the present experiment. Comparison of these animals for weeks 4 through 6 with the late exposure group indicates no

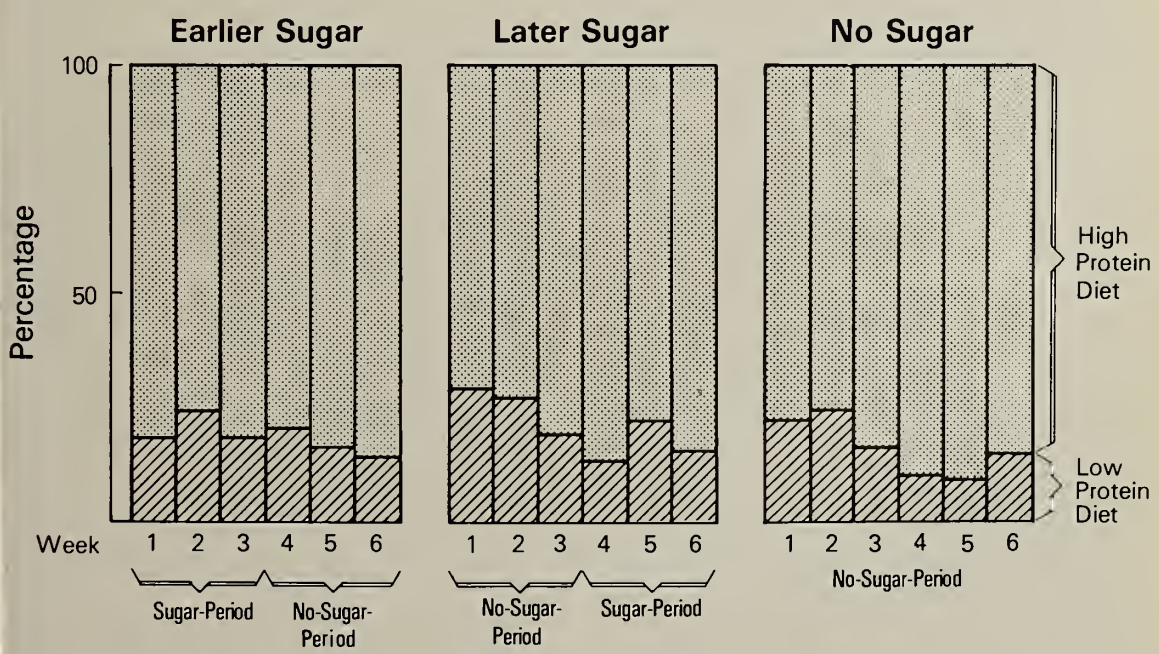


Figure 18–3. Intake of high and low protein diet during six post-weaning weeks in which differential exposure to sugar was provided to three groups of rats.

TABLE 18–XII

SUGAR CONSUMPTION OF RATS UNDER VARIOUS CONDITIONS OF POST-WEANING ACCESS

Access to sugar solutions	*Post-weaning age*			
	1–3 weeks		*4–6 weeks*	
	Grams consumed			
	Sugar	Diet	Sugar	Diet
First 3 weeks	50.6	176.0	...	290.0
Second 3 weeks	...	229.0	97.0	215.0
Never	...	231.0	...	290.0
	Percentage total intake			
First 3 weeks	22	78	...	100
Second 3 weeks	...	100	30	70
Continuous [a]	23	77	30	70

[a] data from previous study in which animals were maintained on high protein diet [31]

differences due to earlier versus later introduction of the sugar solution. Specifically, sugar provides 30 per cent of total intake between the 4th and 6th weeks regardless of whether sugar had been available during the first three postweaning weeks.

From this result it is inferred that the time of introduction of sugar to the young does not necessarily have much influence upon the later craving of sugar, when freely fed. However, the total amount of sugar consumed, of course, is less in the later beginner than in the early beginner.

Chocolate

I have used pure sucrose solution as a source of sweet in the previous experiments. The sweet in this study is chocolate, the same sweet item that is a problem to human beings as shown in Tables 18–VI, –VII, and –VIII. Chocolate is always the child's favorite [20, 25, 35, 41] in spite of the mother's concern because of its caffeine, theobromine, and high sugar content.

In the present study we compare two types of commercially available chocolate with respect to their effect on the health and development of young rats.

For nine weeks, newly weaned rats are offered a basal diet and water along with one of two types of chocolates sold at the market. "Deluxe" chocolate is sweeter than "black" which in comparison is somewhat bitter. The basal diet has the same make up as the high protein diet employed earlier (See Table 18–XI). Deluxe and black chocolate both contain nearly fifty per cent sugar (carbohydrate). The results were compared with those of previous experiments on sugar preferential behavior,[31] as the experimental conditions are similar, except that chocolate is used instead of a sugar solution.

Chocolate consumption gradually increased from about one gram at the first post-weaning week to four to five grams by the ninth week. This is true of deluxe as well as black chocolate. These levels are about the same as in the previous experiment with sugar

solution.[31] Consumption of the basal diet by rats offered either form of chocolate resembles that of rats offered sugar. Consumption of the basal diet increases week by week for the first several weeks and then declines, although it reaches the peak a little earlier and draws a rather long rough plateau when chocolate rather than sugar is available. (See Figure 18–4) Energy coming from deluxe, black or sugar (previous experiment on sugar solution) is roughly 1900, 1600 and 1400 Cal. through the whole experimental period. The sweet supplement provides 41,36, and 34 per cent of the total caloric intake since total intakes are 4600, 4500 and 4100 Cal., respectively. It seems that availability of chocolate reduces the intake of diet to approximately the same level as the availability of sugar.

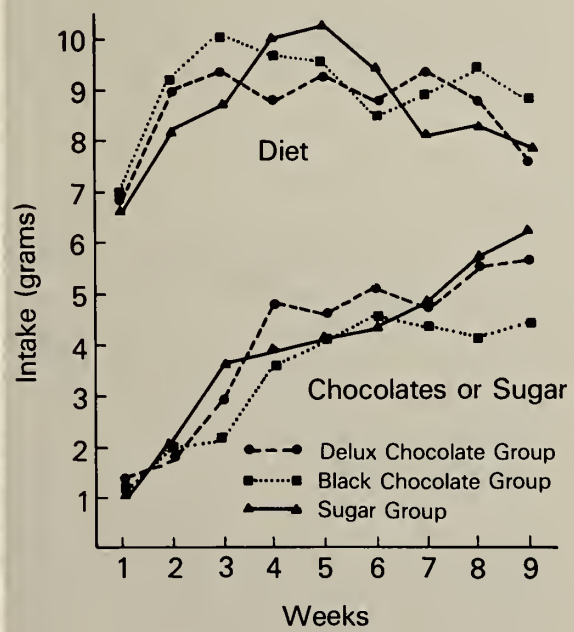


Figure 18–4. The intake of diet and of chocolate or sugar for nine post-weaning weeks during which different groups had access to either of two grades of chocolate or to a water solution of sugar.

However, the caloric intake from chocolate, for the animal with access to chocolate, is greater than the caloric intake from sugar, for the animal with access to sugar.

It is interesting to note that the total caloric intake of the "deluxe" group and the "black" group is about the same, in spite of a larger intake of deluxe chocolate which is sweeter than black. The black group receives fewer calories from chocolate but eats more of the basal diet. After the seventh week of the experiment, in contrast to the preceding weeks, the intake of diet by the black group is a little larger than that of the deluxe group. There is no clue, at present, as to why this occurs.

The final body weight is a little larger in the deluxe group which may be a result of a larger deposition of body fat. The liver weights in each chocolate group are a little smaller and the liver fat content a little higher than those of the sugar group, though all are within the normal range. See Table 18–XIII.

The weight of the spleen and its proportion to the body weight in the chocolate groups are very small compared with the values obtained with the sugar group. This finding, for which we have no clear explanation, has since been replicated in another study of chocolate (Muto, S. and Miyahara, C., unpublished) and requires further investigation.

No differences could be detected in behavior, hair, or general health condition between groups receiving chocolate and sugar.

Two phenomena of these animal experiments are of particular interest in relation to sweet preference. First, in spite of the decline of diet intake

TABLE 18–XIII

BODY AND ORGAN WEIGHTS OF RATS EATING DIFFERENT SWEETS

Sweet	*Whole Body*	*Liver*		*Spleen*	*Kidney*	*Fat Deposit* [a]
	Weight (gm)					
Deluxe chocolate	366	12.2	(4.8) [b]	0.63	2.34	18.9
Black chocolate	326	11.3	(4.4)	0.58	2.21	14.6
Sugar	322	15.4	(3.9)	1.03	2.30	14.9
	Percentage of body weight					
Deluxe chocolate		3.36		0.17	0.64	5.16
Black chocolate		3.44		0.18	0.67	4.48
Sugar		4.76		0.33	0.70	4.63

[a] deposits around kidneys and testes and beneath skin of abdomen
[b] figure in parenthesis is the weight of liver fat as a percentage of total liver

after several weeks, the consumption of sugar or chocolate continues to increase and, at the end of the experimental period, accounts for nearly 50 per cent of total caloric intake. Thus, even after the normal appetite for the basal diet declines, presumably as a function of the slowdown of growth velocity, the appetite for items that taste sweet does not subside. Second, the rats never neglected the basal diet even in the midst of a liberal supply of chocolate or sugar solution. It may be that some instinctual mechanism has evolved that causes the rat to like and to continue eating the basal diet. On the other hand, the taste variety that comes from eating both the diet and sweet items may be the controlling factor.

CONCLUSIONS

Finally, I would like to add a few more general words concerning preferential behavior for food of young individuals. I feel that apart from the effects of transient or momentary changes in physical, emotional, or environmental conditions, there are three fundamental factors controlling food intake. They change with growth process according to individual patterns, but still are common to all individuals.

One factor controlling food intake may be the body's changing need for nutrients. Needs change with the maturation of various tissues and organs as shown beautifully in the performance of Richter's rats. I have tentatively called this factor the age specificity in nutrition, in discussing our previous experiment of calcium deficiency in growing rats.[19]

Another diet control factor is the progressive change in taste sensibility as a function of the cultural pattern under which our daily life is structured. If I could understand these factors more clearly, it would be possible to interpret the finicky eating behavior of the child more satisfactorily and provide more appropriate guidance in how to raise children.

A third factor which I have called "taste individuality" may be critical. We have repeatedly observed a few animals that are exceptional in their sweet preference behavior. They do not consume as much sugar as the rest of the animals in their group even when sugar is available in generous supply. They are at an advantage since they do not develop fatty livers, as the others do.[31] Yet the cause of their response to sugar is a mystery. More over, nothing is known about the later development of preferential behavior for sweet or other tastes in these individuals. However, we need to pay attention to such unusual individuals to advance our understanding of development.

These studies are a mere start of the goal I have been hoping to reach.

REFERENCES

1. Adolf, E.F.: Urges to eat and drink in rats. *Amer. J. Physiol.* 151: 110–125, 1947.
2. Barnett, S.A. and Spencer, M.M.: Experiments on the food preferences of wild rats. *J. Hygene;* 51: 16–34, 1953.
3. Bines, J.A., Suzuki, S. and Balch, C.C.: The quantitative significance of long-term regulation of food intake in the cow. *Brit. J. Nutr.,* 23: 695–704, 1969.
4. Committee on Nutrition: Factors affecting food intake. *Pediatrics.* 33: 135–143, 1964.
5. Davis, C.M.: Self selection of diet by newly weaned infants. *Amer. J. Dis. Child.,* 36: 651–679, 1927.
6. Dickens, D.: Factors related to food preferences. *J. Home Economics,* 57: 427–430, 1965.
7. Hamilton, C.L.: Physiologic control of food intake. *J. Amer. Diet. Assn.,* 62: 35–40, 1973.
8. Harris, L. E. and Chan, J.C.M.: Infant feeding practices, *Amer. J. Dis. Child.,* 117: 483–491, 1969.
9. Hoebel, B.G.: Feeding: Neural control of intake. *Ann. Rev. Physiol.,* 33: 533–568, 1971.
10. Krauss, R.M. and Mayer, J.: Autoregulation of intake of amino-acids; Reversal of an unwise dietary preference in rats under conditions favorable for learning. *Nature,* 202: 463–464, 1964.
11. Kupfermann, I.: Eating behavior induced by sounds. *Nature,* 201:324, 1964.
12. Macy, I.G.: *Nutrition and Chemical Growth in Childhood,* Vol. I Evaluation. Springfield, Charles C. Thomas, 1942, pp. 84.
13. Mayer, J.: Regulation of food intake. In Beaton, G.M. and McHenry, E.W. (Eds): *Nutrition: A Comprehensive Treatise,* New York, Academic Press, 1964, pp. 1–40.
14. McCleary, R.A.: Taste and post-ingestion factors in specific hunger behavior. *J. Comp. Physiol. Psychol.,* 46:411–421, 1953.
15. Menaker, L. and Navis, J.M.: Appetite regulation in the rat under various physiological conditions: The role of dietary protein and calories. *J. Nutr.,* 103:347–352, 1973.
16. Murata, H., Takahashi, M., Muto, S. and Naito, J.: Dietetic studies on children's likes and dislikes, poor appetite and obesity; II. Taste for foods classified into eleven groups. Report of the Studies of Nippon Aiiku Research Institue, No. 5: 153–157, 1970. (in Japanese with English abstract).
17. Muto, S.: A trial of using pupea as protein source for the weaning baby. *Jap. Med. J.,* (Nihon Iji Shinpo), 1156: 1261–1262, 1944. (in Japanese).
18. Muto, S.: Steamed extracts of green leaves as vitamin C sources for the infant, *Jap. Med. J.,* 1142:894–895, 19 (in Japanese)

19. Muto, S. and Akazawa, N.: The effect and the age difference of the Ca deficiency and its replinishment on the growth and the bone composition of the young animal. I. The influence of body weight, height, bone weight and the ash, Ca, and P contents of the bone. *J. Jap. Soc. Food and Nutr.,* 20: 482–486, 1968. (in Japanese with English summary) II. Relationship of bone composition, serum alkaliphosphatase, X ray picture of the bone and food intake with Ca deficiency and its replinishment, *ibidem* 20:487–491, 1968. (in Japanese with English summary).
20. Muto, S., Hara, M. and Tsuda, R.: Sweets, spices and drinks in the dietary in early childhood. *J. of Child Health,* 23: 86–89, 1965. (in Japanese)
21. Muto, S., Hoshino, K., Arai, M., and Hara, M.: The nutrition survey of the nursery school children; I. Data on clinical and biochemical findings. *J. Jap. Soc. of Food and Nutr.,* 9:67–72, 1956.
22. Muto, S., Hoshino, K., Arai, M., and Hara, M.: The nutrition survey of the nursery school children. II. Nutrient and food intakes. ibid, 9: 131–139, 1957. (in Japanese with English summary)
23. Muto, S. and Iwasaki, S.: An experiment of using fish meal in place of egg and fresh fish for the weaning baby. *J. Child Health,* 9:34–38, 1941 (in Japanese)
24. Muto, S., Kato, M. and Kuwabara, K.: Study on the cheap sweets shop. *Jap. J. Home Economics Dept.,* 5:1–5, 1968 (in Japanese)
25. Muto, S., Kato, M. and Takagami, H.: A study on the between meal of the children under care of institution. ibid, 7:1–6, 1960. (in Japanese)
26. Muto, S. and Kobayashi, Y.: Poor appetite of the baby and the child, I. Survey of its occurrence and course, *Jap. J. Pediat.,* 17:1178–1181, 1964. (in Japanese)
27. Muto, S. and Kobayashi, Y.: Poor appetite of the baby and the child. II. Survey of their growth, environment and dietary history, ibid. 17:1315–1318, 1964 (in Japanese)
28. Muto, S. and Matsushima, T., Kobayashi, Y. and Kashiwabara, S.: Nutritional and dietary study on young children. II. Nutrient intakes in general and growth and health conditions, *J. Jap. Soc. of Food and Nutr.,* 14:218–223, 1969 (in Japanese with English abstract).
29. Muto, S. and Miyahara, C.: Eating behavior of the kindergarten and the school children. I. Incident of poor appetite in various age groups of healthy children. *Jap. J. Home Economics,* 20:59–63, 1969 (in Japanese with English abstract).
30. Muto, S. and Miyahara, C.: Eating behavior of the kindergarten and the school children. II. Relationship of poor appetite with growth and health condition. ibid. 20:64–68, 1969 (in Japanese with English abstract).
31. Muto, S. and Miyahara, C.: Eating behavior of young rats: experiment on selective feeding on diet and sugar solution. *Brit. J. Nutr.* 28:327–337, 1972.
32. Muto, S., Mizuno, K. and Kobayashi, Y.: Dietary patterns of Japanese and American preschool children in Tokyo, *J. Amer. Diet. Ass'n.,* 55:252–256, 1969.
33. Muto, S., Takahashi, M. and Konuma, Y.: Study on soybean products as the main protein source of solid food for the weaning baby: method of preparation and cooking. *J. Child Health,* 21:37–49, 1962 (in Japanese)
34. Muto, S., Takahashi, M., and Konuma, Y.: Soybean products as protein sources for weanling infant. *J. Amer. Diet. Ass'n.,* 43:451–456, 1963.
35. Naito, J., Muto, S., and Yamanouchi, A.: Investigation into the maternal and child health in the danch (the place where a large number of appartment houses are grouped together); Part 3. Report of the Studies of Nippon Aiiku Research Institute; No. 3:6–11, 1967. (in Japanese)
36. Powley, T.L. and Keesey, R. E.: Relationship of body weight to the lateral hypothalamic feeding syndrome. *J. Comp. Physiol. Psychol.,* 70:25–36, 1970.

37. Richter, C.P. and Barelare, B.: Nutritional requirement of pregnant and lactating rats studied by the self-selection method. *Endocrinology,* 23: 15–23, 1938.
38. Richter, C.P., Holt, L.E., and Barelare, B.: Nutritional requirement for normal growth in rats studied by the self-selection method. *Amer. J. Physiol.,* 122:734–744, 1938.
39. Scott, E.M.: Self selection of diet; I. Selection of purified components, *J. Nutr.,* 31:397–406, 1946.
40. Sweet, C.: Voluntary food habits of normal children. *Journal of the American Medical Association,* 107:765–768, 1936.
41. Takahashi, M., Murata, H., Muto, S., and Naito, J.: Dietetic studies on children's likes and dislikes, poor appetite and obesity. I. stature, health and eating habits of children and their mother's attitude toward feeding. Report of the Studies of Nippon Aiiku Research Institute, No. 5:141–152, 1970. (in Japanese with English abstract).
42. Young, P.T. and Chaplin, J.P.: Studies of food preference, appetite and dietary habit. III. Palatability and appetite in relation to bodily need. *Comp. Psychol. Monog.,* 18:1–43, 1945.

PART V
RELATING TASTE TO INGESTION

Chapter 19

THE ROLE OF SWEETNESS IN THE FOOD PREFERENCE OF YOUNG ADULTS

Herbert L. Meiselman

As a ten year veteran of taste research, it has always surprised me how so much talk can be heard about taste, in this context sweet taste, with little or no mention of food as we adult humans know it. Laboratory studies of taste, including sweetness, have consistently used supposedly simple, supposedly well understood stimuli like sucrose and saccharin, and with few exceptions [3,4] have avoided the world of food which is the natural vehicle for sweetness and other tastes. When I was first approached as a potential contributor to this conference, it was within the area of taste psychophysics. Instead, with the interest and support of the conference planners, I have decided to attempt to place the concept of sweetness within the world of food behavior.

The area which I have chosen to address is that of food preference judgements. I will be concerned with what people say they like to eat. The data will be limited to the food preferences judgments of young male adults. An understanding of the food preferences of the population and of specific subgroups can identify the medium for exposure to sweet and ingestion of sweet materials. The assumption that sweetness is encountered only through "sweets" is probably unwarranted, and more objective information is needed on what foods make up the preferred sweet repertoire. I have chosen two examples of food preference analysis to explore the possible role of sweetness in food preference behavior. They are the food preferences of blacks as compared with whites, and the food preferences of overweights as compared with underweights and normals. These two were chosen because of their relevance to social and medical issues.

THE FOOD PREFERENCE SURVEY

Our food preference data come from surveys administered to military personnel in groups.[1] The military has long pioneered in the development of food preference and acceptance measurement techniques, and in the acquisition of food attitudinal data.[5] Our present survey format,[1]

Acknowledgment: I thank Toby Reed for his aid in analysis of the data.

printed on mark-sense sheets for automated data reduction, is a list of 378 food names, listed randomly. Included are members of all food classes (appetizers, soups, entrees, vegetables, starches, desserts, etc.). Three questions are asked concerning each food item in the survey: have you tried it, how much do you like or dislike it and how often do you want to eat it in days per month. If you have never tried the food, you go on to the next item; if you have tried the food, you assign a hedonic rating for how much you like it, and a preferred frequency rating for how many days per month you want it served. The hedonic rating scale is a nine point category scale (1, 2, 3, . . ., 9) with a neutral point at 5. This scale was developed at the Quartermaster Food & Container Institute [5] and is used throughout the food industry. The frequency scale is our own invention; one of several we have tried. This one uses a two digit system to express the number of days per month a food is desired, from 00, 01, . . ., 30. These food preference scales reflect what people say they like to eat. We have devoted considerable effort to trying to correlate these attitudinal measures to behavioral data on what people actually do in a real eating situation.

The first two pages of the survey form request demographic information, on age, race, sex, education level, weight, height, city size where raised, state where raised, cooking type raised on, and preferred food types. Clearly, the number of demographic analyses possible approaches infinity when one considers that our sample size is about 4000, making most cells in the data set large enough for statistical analysis.

Overall food preferences

In order to provide a perspective for the data, some indication of the place of sweet foods within high preference foods is given in Table 19–I. For this table, we obtained ratings on the 378 foods on both the hedonic and preferred frequency measurement scales. The top fifty foods on each scale were then divided into food classes. About 40% of the top fifty foods are sweet, but only 7/50 for the hedonic scale and 3/50 for the frequency scale are desserts. Fresh fruit shows a high frequency of occurence within the top fifty for both scales, whereas sweet beverages and desserts appear more dependent on scale type. This suggests that the common tendency of looking for sweetness solely within the dessert class might be incorrect. The number of sweet foods within the 50 foods rated at the bottom of the 378 foods is smaller than within the top 50 but not unimportant, 7/50 for the hedonic scale data and 9/50 for the frequency scale data. Both sets include fruits, beverages, and desserts made with unpopular items (raisins, prunes, etc.).

Food preferences of Blacks

Our analysis of food preferences of blacks has proceeded through several

TABLE 19–I

SWEET FOODS IN THE TOP 50 (OUT OF 378) FOODS

	HEDONIC	*PREFERRED FREQUENCY*
Sweet Beverages	3	10
Desserts	7	3
Fresh Fruits	10	9
Total	10	22

stages on the way to what we feel is a highly conservative list of black food preferences which are significantly (in the statistical sense) different from white food preferences. The first analysis of variance examined differences between the three main subject groups listed on the survey, Caucasian, Negro, and Oriental; this resulted in four lists of food judgments relevant to this discusssion. These are, a list of foods with significantly higher hedonic ratings by blacks than whites, a list with significantly higher preferred frequency ratings by blacks than whites, a list with significantly higher hedonic ratings by whites than blacks, and one with higher preferred frequency ratings by whites than blacks. The preferred frequency lists generally were longer, containing more differentiated items than the hedonic list. That is, desired frequency of serving produced more discriminations than hedonic score. These lists confirm many of the popularized distinctions between American food ("white" food) and Afro American food (black or soul food), such as higher preferences by blacks for pork products, rice, and green vegetables and higher preference by whites for beef and white potato products.

Additional analyses were performed to clean the data of the single most potent source of artifact, the possible confounding between black food preferences and food preferences of southerners, both black and white. From our demographic data, a southerner can be identified in at least two ways. One criterion for identifying a person as a southerner was as a person raised in one of eleven southern states; the other, as a person raised on southern style food. We did analyses using both criteria and found that both criteria gave the same results. The analysis presented below used the criterion of state where raised. Four groups were formed, black southerners, black northerners, white northerners, and white southerners. Analyses of variance again produced the four hedonic and frequency food preference lists mentioned above for black preferences and for white preferences. Our criterion of a black food preference was tri-partite. We required that it show a statistically significant difference from the white rating for southern blacks and southern whites, for northern blacks and northern whites, and for overall black and white (the analysis mentioned above). This highly conservative listing eliminates those black food preferences which are limited to the north or south, or which are borderline and hence do not reach statistical significance 3 times. Tables 19–II, III, IV and V list the black food preferences, and Table 19–VI lists the white preferences according to the criteria.

Table 19–II lists the entree or main dish items, giving the mean hedonic ratings for blacks and whites. On the far right are additional notes indicating which subgroups significantly differ from the simple effect described by the other data. *B* and *W* represent black and white respectively, and *N* and *S* represent north and south. A minus (−) sign indicates a lowered preference and a plus (+) indicates higher preference. Thus, barbecued spareribs are preferred by blacks over whites according to the conservative criteria described above, and also, white northerners (WN) have lower ratings (WN−) than white southerns.

TABLE 19–II

ENTREE PREFERENCES OF BLACKS

ENTREES	*HEDONIC*		*FREQUENCY*		
	B	*W*	*B*	*W*	*NOTES*
barbecued beef cubes	6.56	6.03	10.03	6.95	
barbecued spareribs	7.65	6.68	15.31	9.41	WN–
boiled pigs' feet	6.64	3.49	10.09	3.20	BS+
braised liver w/onions	5.44	4.47	7.65	5.16	
chitterlings	7.13	3.59	12.78	4.04	WN–
fried fish	6.84	6.31	12.16	8.78	WN–
grilled bologna	5.57	4.90	8.34	5.97	
pepper steak	6.94	6.53	11.84	9.08	
pickled pigs' feet	6.54	4.25	11.88	4.92	BS+
pork chop suey	6.26	5.25	10.56	6.79	BS+
pork hocks	6.57	4.85	11.44	5.72	WN–
salmon	6.18	5.69	9.34	7.12	
sausage links	7.07	6.68	15.09	13.47	
stuffed cabbage	5.85	5.31	8.88	6.56	
tuna salad sandwich	6.72	6.36	11.80	9.35	

Therefore, in addition to a black-white difference there is an interaction of race x region. Notice the large number of pork products, and the several fish products. The 378 foods are divided into 33 food classes; the class of fish and fish products showed an overall significant effect between whites and blacks. However, the broader entree class did not, because many of the items, largely the non-pork items, were not differentially preferred.

Table 19–III shows starch and vegetable items; notice the lack of white potato items and the presence of rice items and green vegetables especially lima beans. The class of green vegetables showed an overall significant effect. Table 19–IV, desserts, does not have many items, although there are 84 desserts in our 378 foods. The analyses which were combined for this final list all showed many pineapple and dried fruit items, some of which remain on this final listing. The

TABLE 19–III

VEGETABLE AND STARCH PREFERENCES OF BLACKS

STARCH/VEG	*HEDONIC*		*FREQUENCY*		
	B	*W*	*B*	*W*	*NOTES*
baked macaroni & cheese	6.94	6.46	12.71	9.17	
buttered mixed vegetables	6.24	5.90	11.73	10.14	
buttered succotash	5.56	4.99	8.36	5.81	
cabbage	6.31	5.12	10.53	6.58	WN–
canned lima beans	5.81	4.72	9.30	5.65	WN–
carrot raisin celery salad	4.78	4.29	7.19	4.97	
collard greens	7.17	4.81	14.25	6.26	WN–
fried cabbage	5.78	4.67	8.85	5.39	
fried rice	6.39	5.66	10.81	7.70	
frozen lima beans	5.83	4.50	9.34	5.76	
hominy grits	6.55	5.10	14.57	7.77	WN–
mustard greens	6.33	4.45	11.06	5.24	WN–
steamed rice	6.53	5.47	12.91	7.89	WN–
sweet potatoes	6.87	5.31	11.89	6.76	WN–
turnip greens	6.63	4.36	11.61	5.46	WN–

TABLE 19–IV

DESSERT PREFERENCES OF BLACKS

DESSERTS	*HEDONIC*		*FREQUENCY*		
	B	*W*	*B*	*W*	*NOTES*
bread pudding	5.76	5.14	8.95	5.92	
pineapple sundae	6.37	5.83	10.49	8.12	
raisin cookies	5.78	5.29	9.18	7.01	
raisin pie	4.89	4.42	7.58	4.58	
sweet potato pie	7.36	4.53	14.60	5.23	WN−
vanilla wafers	6.20	5.73	10.30	7.73	
yellow cake	5.85	5.57	8.57	6.50	

classes of cookies, cakes, and pies all showed significant preferences by blacks.

I have saved the most interesting for last, Table 19–V , for beverages. Even with the conservative criteria for inclusion as a black food preference, the large listing shown emerged. There is a heavy concentration of fruit flavored juices, sodas, and non-carbonated drinks. The class of fruit drinks shows a highly significant difference in preference by blacks as compared with whites. The point to be made, in the context of this conference, is that the food class in which there appears to be major differential preference between blacks and whites related to sweetness appears to be fruit drinks. It can be predicted that the food ingestion behavior of blacks would reflect these preference data, and that, therefore, the food class which would account for differential exposure of blacks to sweetness would be fruit beverages.

The last table (Table 19–VI) in this series shows the cases of significantly greater food preferences of whites, with an attached listing of food group-

TABLE 19–V

BEVERAGE PREFERENCES OF BLACKS

BEVERAGES	*HEDONIC*		*FREQUENCY*		
	B	*W*	*B*	*W*	*NOTES*
cherry flavored drink	6.36	5.55	13.40	8.72	WN+
cherry soda	6.38	5.71	12.87	9.26	WN+
fruit punch	6.84	6.03	14.69	9.69	WN+
gingerale	6.48	5.93	13.06	10.30	WN+
grape flavored drink	6.84	5.91	15.41	10.08	
grape juice	6.95	6.24	16.32	12.94	
grape lemonade	6.50	5.50	13.70	8.92	
grape soda	6.92	5.77	16.04	9.75	
grapefruit juice	6.41	5.66	13.90	10.54	
grapefruit orange juice	6.81	6.12	15.98	12.97	
grapefruit pineapple juice	6.46	5.57	13.96	10.05	
lemon lime soda	6.32	5.68	13.66	10.78	WN+
lemonade	7.21	6.73	16.17	13.00	
lime flavored drink	5.91	5.28	11.06	8.09	WN+
orange flavored drink	6.92	6.04	16.20	10.29	
orange soda	7.14	6.04	16.97	11.34	
pineapple juice	6.37	5.69	13.32	9.23	
prune juice	4.59	3.97	7.30	4.41	

TABLE 19–VI

FOOD PREFERENCES OF WHITES

ITEMS	*HEDONIC*		*FREQUENCY*		*NOTES*
	B	*W*	*B*	*W*	
celery & carrot sticks	5.01	5.80	8.07	12.05	WN+
fresh coffee	5.60	6.57	15.42	19.92	
hashed brown potatoes	6.59	7.16	12.75	14.50	
milk	7.60	8.08	22.09	25.10	
tomato juice	4.97	5.85	9.25	12.44	
tossed green salad	6.51	7.15	14.31	17.73	WN+
FOOD CATEGORIES PREFERRED ON HEDONIC SCALE ONLY					
chocolate desserts	non-green buttered vegetables				Mexican food
butterscotch desserts	white potatoes				Italian food

ings preferred on the hedonic scale only. It appears that the area of chocolate desserts is one in which the differential exposure to sweetness might be exercised for the whites as compared with blacks. One interesting point is the reduced preference for milk by blacks.

At the present time, it is unclear why the number of foods significantly preferred by blacks is greater than the number preferred by whites. It may be related to the relative unavailability of the black foods in the military food system.

Food preferences of relative weight groups

Our attempts to deal with the complicated dimension of relative body size have been characterized by the fumblings and errors of the novice. We ask your patience with our approaches. The raw data with which we had to work were the approximately 4000 food preference surveys with the height and weight provided by each survey respondent. Our first approach was to calculate the somatotype ponderal index (SPI) from height divided by the cube root of weight.[6, 7] The distribution of SPI was then cut at either one or two standard deviations to produce groups of overweight (low SPI), and underweight (high SPI). The hedonic scale and preferred frequency scale data were analyzed by analyses of variance to determine whether significant differences existed for each of the 378 foods. The number of significant differences within several large food classes are shown in Table 19–VII. H—indicates a higher preference by the underweight subjects and H+ indicates a higher preference by the overweight subjects. Examination of these data initially led us to the conclusion that the hedonic scale analysis did not yield many differences between overweight and underweight, and did not isolate any food class which was differentially affected by the weight variable. However, the frequency scale indicated a dramatic preference for desserts by the underweight subjects. The appearance of a "dessert phenomenon" when one is talking about sweetness and obesity is always welcome. We were not prepared, however, for higher preference to be shown by the underweight group. Fortunately we did not stop the search at this point.

A second set of analyses was run on

TABLE 19–VII

NUMBER OF FOODS SHOWING SIGNIFICANT DIFFERENCES IN PREFERENCE BETWEEN OVERWEIGHT AND UNDERWEIGHT

		PREF. FREQ.	*HED.*	*Class No.*
dessert	H−	49	7	84
	H+	0	0	
beverage (sweet)	H−	2	0	31
	H+	0	1	
beverage (unsweet)	H−	2	1	29
	H+	1	5	
entree	H−	8	1	111
	H+	0	8	
starch	H−	6	1	30
	H+	0	1	
vegetable	H−	3	0	64
	H+	0	7	
fruit	H−	12	1	29
	H+	0	0	
misc	H−	0	0	8
	H+	0	0	

H+ = preferred by heavier individuals
H− = preferred by lighter individuals
overweight = +2 standard deviations SPI (HT/$\sqrt[3]{WT}$)
underweight = −2 standard deviations SPI (HT/$\sqrt[3]{WT}$.)

the hedonic and preferred frequency data, this time using a one standard deviation measure for overweight and underweight, and dividing the population into four groups. The group above one standard deviation was the overweight group, and the group between the sample mean and the plus one standard deviation group was referred to as "slightly overweight." Similarly, underweight and slightly underweight groups were formed. The analyses now had to examine the number of foods (out of 378) which showed statistically significant differences when various combinations of the four groups were compared (Tables 19–VIII and IX).

The impressive number of desserts showing significance in comparisons of the various weight groups is still evident in the table of preferred frequency data. Comparisons between slightly heavy or heavy and slightly light or light, in other words comparisons which cross the mean, yield substantial numbers of desserts for which the lights have higher ratings than the heavies. In the analyses of 33 food classes, several dessert classes (cakes, puddings, ice cream) showed this marked preference. In Table 19–VIII, the numbers of entrees and fruits yielding significance are also not small and the statistically significant class analyses for fresh fruit and sandwiches reflected this. For most food classes note that the largest number of differences are in the comparison of slightly heavy with slightly light, not between the extreme groups.

TABLE 19–VIII

NUMBER OF FOODS SHOWING SIGNIFICANT DIFFERENCES IN PREFERENCE BETWEEN OVERWEIGHT (O), SLIGHTLY OVERWEIGHT (SL.O), SLIGHTLY UNDERWEIGHT (SL.U) AND UNDERWEIGHT (U)

		PREFERRED FREQUENCY					
		SL.O vs O	*SL.U vs O*	*O vs U*	*SL.U vs SL.O*	*SL.O vs U*	*SL.U vs U*
dessert	H−	0	18	6	67	61	20
	H+	0	0	0	0	0	0
beverage (sweet)	H−	0	2	2	8	4	2
	H+	0	0	0	0	0	0
beverage (unsweet)	H−	0	2	1	3	3	2
	H+	2	2	1	1	0	0
entree	H−	0	4	9	17	16	11
	H+	1	3	0	0	0	0
starch	H−	0	2	5	10	11	4
	H+	0	0	0	0	0	0
vegetable	H−	0	0	1	1	2	1
	H+	0	0	0	0	0	0
fruit	H−	0	6	12	16	11	4
	H+	0	0	0	0	0	0
misc.	H−	0	0	0	0	0	0
	H+	0	1	0	0	0	0

H+ = preferred by heavier individuals
H− = preferred by lighter individuals
Overweight = +1 standard deviation SPI ($HT/\sqrt[3]{WT}$)
Slightly Overweight = between mean and plus one S. D.
Slightly Underweight = between mean and minus one S. D.
Underweight = −1 standard deviation SPI

The hedonic data (Table 19–IX) show the phenomenon of increased stated preference for desserts by those on the light side of the SPI distribution. However, there has been a substantial increase in the number of foods significantly preferred by those on the heavy side of SPI distribution, especially for entrees. Both vegetables and starches also show increased numbers of items preferred by overweight over underweight. In the food class analyses, none of these effects seen with food item analysis appeared. Returning to the previous Table 19–VII for heavy vs light, the two group comparsison, it can be noted that both entrees and vegetables did show some foods preferred by heavy over light subjects.

Thus, the data using an SPI measure of overweight and underweight and both an hedonic and preferred frequency measure of food preference do not present a simple picture of the relationship between body size and food preference. Lighter young men prefer desserts with greater frequency of serving than heavier young men, and like them more. Heavier young men like entree items more than lighter young men, but do not want them served more often.

Rather than stopping at this point,

TABLE 19–IX

NUMBER OF FOODS SHOWING SIGNIFICANT DIFFERENCES IN PREFERENCE BETWEEN OVERWEIGHT (O), SLIGHTLY OVERWEIGHT (SL.O), SLIGHTLY UNDERWEIGHT (SL.U), AND UNDERWEIGHT (U)

		HEDONIC					
		SL.O vs O	*SL.U vs O*	*U vs O*	*SL.U vs O*	*SL.U vs SL.O*	*SL.U vs U*
dessert	H−	5	22	40	19	37	15
	H+	0	0	0	0	0	0
beverage (sweet)	H−	1	6	6	3	4	2
	H+	1	1	0	0	0	0
beverage (unsweet)	H−	0	0	1	0	1	1
	H+	6	8	5	3	2	0
entree	H−	0	1	2	1	2	3
	H+	16	36	18	19	3	0
starch	H−	1	3	8	3	6	3
	H+	3	7	3	5	1	0
vegetable	H−	0	0	0	0	0	0
	H+	3	14	6	9	1	0
fruit	H−	1	10	12	4	6	3
	H+	0	0	0	0	0	0
misc	H−	0	0	0	0	0	1
	H+	0	1	0	1	0	0

H+ = preferred by heavier individuals
H− = preferred by lighter individuals
overweight = +1 standard deviation SPI (= HT/$\sqrt[3]{WT}$)
slightly overweight = between mean and plus one S.D.
slightly underweight = between mean and minus one S.D.
underweight = −1 standard deviation SPI

we decided to relate our data to the other data in the obesity literature which uses a different measure of body size, one related to the Metropolitan Life Tables.[2] Using plus fifteen percent as a measure of overweight and minus fifteen percent as a measure of underweight, we again ran an analysis of variance on the 378 food items with the light, normal and heavy groups. The number of subjects falling in each group is interesting; for most foods the sizes of the three groups were approximately 100, 2000, and 500 indicating that five times as many people qualified as being overweight as underweight according to these criteria. For relatively unfamiliar foods, these numbers were smaller because some subjects had indicated they had never tried the item. This is yet another reflection of the general obesity problem which has received so much attention.

Table 19–X presents the number of foods within each food class which show a statistically significant difference in preference (hedonic or preferred frequency). For both frequency and hedonic scales the dessert phenomenon described above for the data based on the distribution of SPI

TABLE 19–X

NUMBER OF FOODS SHOWING SIGNIFICANT DIFFERENCES IN PREFERENCE BETWEEN OVERWEIGHT (O), NORMAL (N), AND UNDERWEIGHT (U)

		PREFERRED FREQUENCY			*HEDONIC*			
		U vs N	*U vs O*	*N vs O*	*U vs N*	*U vs O*	*N vs O*	*Class No.*
dessert	H+	0	0	1	1	0	7	84
	H−	5	6	2	0	0	0	
beverage (sweet)	H+	0	0	1	0	1	0	31
	H−	0	0	0	0	0	0	
beverage (unsweet)	H+	0	2	2	4	5	4	29
	H−	1	0	2	0	0	0	
entree	H+	0	0	10	11	37	46	111
	H−	3	2	1	0	0	0	
starch	H+	0	0	0	1	2	6	30
	H−	2	3	1	1	0	0	
vegetable	H+	1	0	1	1	3	7	64
	H−	3	1	0	2	2	0	
fruit	H+	0	0	0	3	3	0	29
	H−	0	0	0	0	0	1	
misc	H+	0	0	0	0	0	0	8
	H−	3	3	0	0	0	0	

H+ = preferred by heavier individuals
H− = preferred by lighter individuals
overweight = +15% on Metropolitan Life Tables
underweight = −15% on Metropolitan Life Tables

scores has disappeared. The popularity of entrees with the heavy part of the sample is very clear, especially for the hedonic scale as was observed with the SPI data above. This again permits one to draw the conclusion that heavy young males prefer entrees more than light or normal weight young males. For sweet foods (desserts, beverages, and fruits) there appears to be no difference in preferences of light, normal, and heavy subjects when body size is described by the Metropolitan Life Tables.[2]

SUMMARY

I have examined the food preferences of approximately 4000 young male personnel in the United States Armed Forces. Their food preferences have been studied using a food preference survey of 378 food items. The survey asks how much they like each item (hedonic scale), and how often they would like each item served (preferred frequency scale). Overall, about 40% of the fifty most popular foods are sweet foods. Special attention was paid the sweet food in contrasting the preferences of blacks with whites, and of overweight, normal, and underweight people. The analysis

of racial food preferences indicates a high preference by blacks for sweet fruit beverages, suggesting that this food class might play a role in differential exposure of blacks to sweetness and in differential ingestion of sweet items. Analyses of food preferences of groups differing in relative body size indicated a more complex situation. It was determined that compared to normal weight subjects, underweight people rated desserts more highly (hedonic scale) and requested both desserts and entrees more frequently (preferred frequency). Overweight people, on the other hand, rated entrees more highly and said they preferred them more frequently. The complexity of the findings is not consistent with any simplistic view of food preferences, relative body size, and the role of sweet foods.

REFERENCES

1. Meiselman, H.L., Symington, L.E., Branch, L., Waterman, D. and Reed, T.: Armed Forces Food Preferences. United States Army Natick Laboratories Technical Report, 1974, 75–63–FSL, Dec. 1974.
2. Metropolitan Life Insurance Company, New Weight Standards for Men and Women, *Statistical Bulletin,* 1959, 40, p. 2.
3. Moskowitz, H.R., Kluter, R.A., Westerling, J. and Jacobs, H.L.: Sugar sweetness and pleasantness: evidence for different psychological laws. *Science,* 184:583–585, 1974.
4. Pangborn, R.M., Ough, C.S. and Chrisp, R.B.: Taste interrelationship of sucrose, tartaric acid, and caffeine in white table wine. *American Journal of Enology and Viticulture,* 15:154–161, 1964.
5. Peryam, D.R., Polemis, B.W., Kamen, J.M. Eindhoven, J. and Pilgrim, F.J.: Food Preferences of Men in the U.S. Armed Forces. Quartermaster Food and Container Institute, 1960, 160.pp.
6. Sheldon, W.H., Stevens, S.S., and Tucker, W.B. *The Varieties of Human Physique,* New York, Harper and Bros., 1940, p. 347.
7. Waller, R.E., Heights and weights of men visiting a public health exhibition. *British Journal Preventive Social Medicine,* 26:180–185, 1972.

DISCUSSION

Morrison: Could it be that overweight people simply like to eat and so prefer the foods which they think they can eat a lot of? Since they think sweets will make them fat, they reject them in favor of foods that they feel freer to eat.

Meiselman: Anecdotally, I can report that the respondents appear to be filling out the survey honestly and spontaneously. They read through it, item by item, saying things like, "Oh, I love it!" If you are suggesting an underlying motivation, then your hypothesis as well as many others are possible.

Engen: The variability of rating seems to me to be relatively small for such a large group. Measures of the average variability might not tell you much, but you might pay special attention to items with extremely high or low variability of ratings.

Meiselman: We have looked at items with different distributions of scores, because these present differ-

ent practical problems for menu planners. For example an item for which the scores are bimodally distributed should be offered to one group but should not be offered to another (e.g. liver). Low variability makes simpler menu solutions possible.

RODIN: The taste rating data presented in my chapter suggest high group variability in preference ratings. This finding became more important than the mean differences between groups.

MEISELMAN: We already have those data. They could be compared easily. Thank you.

GALEF: I was wondering if your respondents might perceive this questionnaire as effecting the choice of food items which would eventually be presented to them? If so the overweight subjects might be expressing a desire not to be presented with food items they know to be of high caloric content. Perhaps it's not that they wouldn't eat the high caloric items if they were available, but that they would rather not be tempted.

MEISELMAN: I would have to disagree with your hypotheses. One of the strongest attitudes which we have to combat in doing surveys within the military is the attitude on the part of the respondents that the survey will have *no effect,* even if there is an effect that the effect will be very far off.

GRINKER: It is possible to draw some conclusions about diet from a comparison of the adipose tissue of obsese and normal weight subjects since the composition of adipose tissue reflects the nature of ingested fat. There are no significant differences between the two groups suggesting that obese individuals eat the same proportion of fats, carbohydrates, etc. as individuals of normal weight.[1,2]

Thus, overall intakes of sweets, etc. would be proportionately equivalent, but the frequency of eating these items might be less regular. The overweight person may abstain from foods considered to be highly caloric and then periodically overindulge. It is also possible that self reports of overeating reflect the response bias of the overweight person. We have patients who report overeating what they think they should. For example, they will report eating steak and potatoes, but in amounts appropriate for several people. Another possiblity is that the notion that overweight people like sweets is erroneous. We certainly have not found an increased preference for sucrose solutions in our experiments. Furthermore, our subjects often tell us that they overeat items like bread. They are getting their carbohydrates, but in another form.

RODIN: We get the same thing. While it may be true that overweight people want to present themselves well and thus say that they do not eat many sweet things, for many this is not simply self-presentation. Our intake data suggest that there are several extreme instances of overeating what they know they should eat. So fifteen eggs and ten pieces of bacon for breakfast is a morning meal because that is seen as less fattening than a piece of cake.

---

[1] Hirsch, J.: Fatty acid patterns in human adipose tissue. In: *The Handbook of Physiology,* Section 6, Adipose Tissue, Washington, American Physiological Society, 1965, pp. 181–187.

[2] Hirsch, J., and Goldrick, B.: Studies on composition and metabolism of adipose tissue in man. In Rodahl, K. (Ed.), *Fat as a Tissue,* New York, McGraw Hill 1964, pp. 314–328.

MEISELMAN: Then why did the overweight group not show a high preference for salads? Our surveys only ask what foods are preferred and in what frequency of serving. There is no way of getting at serving size in these data, so your point about the portion taken is a good one. Also, the data are consistent in not showing a preference by overweights for sweets.

RODIN: I think it is a combination of preference or palatability and lower caloric value. Many high protein entrees are also quite high in calories although many people are not aware of this. Starches as accompaniments or in casseroles also add greatly to the caloric value of a meal. This may be too simple an explanation but it is possible they are overeating high calorie, non-sweet food because they, as almost all Americans, are poorly informed about good nutrition. Or, as was suggested earlier, they may not be telling the whole truth on your questionnaires which suffer from the same problems as any self report measure. One way to check this alternative is to compare self-reported to actually measured height and weight. It would be an indicator of whether they misrepresent data on these questionnaires.

MEISELMAN: Even better, we are collecting data on what people actually eat in dining halls. From these same large samples, we are also collecting preference surveys, so that we will be able to compare what a man does to what he says. We seem to agree on the critical role of entrees for fat people.

COLLIER: I would like to suggest that it may be the case that response bias is not operating here. You are getting the correct data. If you remember our rat data, active rats increase their intake of carbohydrates. What you may be looking at are differences in activity levels within the human population. Your lightweights are running around engaging in all kinds of activities, while, as we all know, the obese are conserving energy, sitting around, not moving very much. Your data, then, express the effect of activity. The lightweight, active individuals, just as they should, shift their preference in the direction of carbohydrates as an energy source.

MEISELMAN: We have some relevant data on the food preferences of men in three different climates collected within a two week period. We tested Air Force personnel in North Dakota at well below zero, central California at temperatures in the range of 45–55° F, and Florida at temperatures of near 80° F. Even with this range of 100° we could find no systematic differences in preference for starches or other classes.

Chapter 20

SENSATIONS, MEASUREMENT AND PLEASANTNESS: CONFESSIONS OF A LATENT INTROSPECTIONIST

Howard R. Moskowitz

In the last two decades human sensory measurement has received increased attention from psychophysicists, psychologists, food scientists, and neurophysiologists. The attempt to use numbers to represent nuances of sensory experience is attractive for a variety of reasons, not the least of which is that it is an attempt to make public a private aspect of human behavior. Even more important, however, is the insight that these studies of sensory phenomena provide concerning how individuals process information from the external world, and what this processing implies about aspects of human behavior, such as feeding and drinking. This paper focuses on three aspects of sensory measurement applied to taste: procedures which are available for assessing sensory and hedonic experience, substantive findings which continue to recur and possible explanations of some of these findings in terms of an "interpretation theory of hedonics."

MEASUREMENT

Nominal scaling

In 1946, S.S. Stevens proposed that measurement could be arranged into a hierarchy of increasingly potent operations.[15] The lowest level of measurement is classification or nominal scaling. In this procedure individuals sort stimuli into various groups, by one or another rule. Early studies of hedonics relied extensively upon nominal scaling. Usually the observer was presented with a stimulus (e.g., a solution of sucrose, a cotton plug saturated with a noxious odorant like asafoetida, etc.) and asked to classify the impression as being pleasing, displeasing or neutral. Rarely was sensory magnitude assessed via nominal scaling, however.

Although the observer never directly assigns numbers to stimuli, when asked to classify his hedonic impression, experimenters (e.g., Engel [4]) often implied that a stimulus found pleasant by more individuals had a higher pleasantness value.

The intensity of hedonic response, the degree of hedonic tone, was incorrectly inferred from the proportion of the population finding a stimulus to be pleasing or displeasing.

Figure 20–1 shows data collected by Engel,[4] who instructed his observers to classify sucrose solutions as liked, disliked or neutral. Although concentration influences the number of individuals who find the taste to be pleasing, no effect on gradations in pleasantness can be inferred.

The attractiveness of the classification procedure is its simplicity. The observer need not do anything more than classify. Even animals may be trained to classify. Unfortunately, the simplicity of the task clouds the interpretation of the results. Since response categories that reflect graded degrees of liking or disliking are not permitted, stimuli which are just mildly pleasing are classified together with stimuli which are much more pleasing. In addition, it is not clear what criteria the observer uses, and whether these criteria are stable across time and across experimental situations.

Ordinal scaling

The second level of the measurement hierarchy is the ordinal scale. Here the observer indicates that he prefers one stimulus to another, or that one stimulus is sensorically stronger (or weaker) than another.

Psychophysical studies of taste preference using ordinal measurement by Chappell [3] suggested that observers have a preference order for the taste of sugar solutions. Sucrose is preferred to both glucose and to fruc-

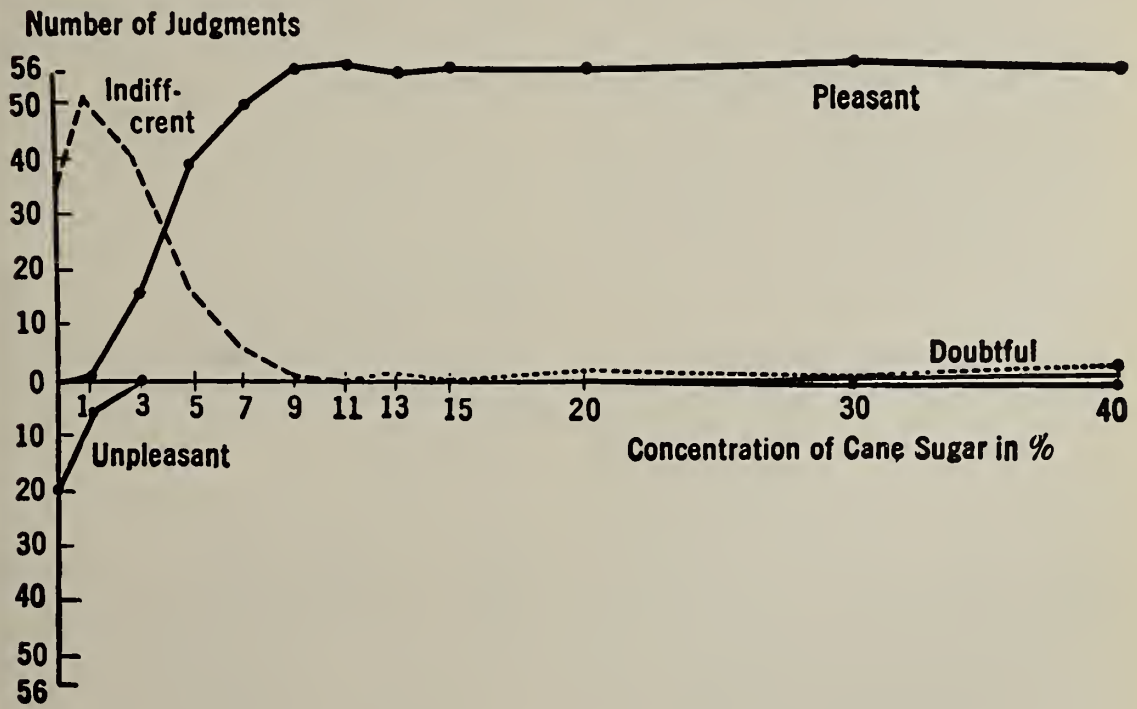


Figure 20–1. Relation between the concentration of sucrose solution (cane sugar) and the proportion of the population stating that they find the taste pleasant, unpleasant or neutral (respectively). Data from Engel (1928), redrafted by Beebe-Center (1932), with the permission of Van-Nostrand Reinhold, Inc.

tose (which are equivalent to each other), and these latter sugars are both preferred to maltose. It is important to know the rank order of preferences, but the ordinal scaling procedure leaves unanswered the question of the degree to which one sugar is preferred to another.

In animal experimentation the experimenter often observes choices between two or more stimuli (e.g., vary ing concentrations of sugar solution). These choices resemble ordinal scaling. Solutions which the animal drinks more of are inferred to be more pleasant than those solutions which the animal drinks less of. However, it is necessary to grant the assumption that the animal drinks only for pleasure to infer the degree to which one solution is preferred over another from such data.

Ordinal scaling, like nominal scaling, may fail to provide an indication of the extent to which one stimulus is preferred to another, a metric for degree of liking. The metric information is obtained from the elegance of the mathematical treatment, not from the behavior of the observer.

Interval scaling

Interval scaling and ratio scaling are higher forms of measurement in which the observer becomes a measuring instrument which generates numbers to reflect response magnitudes. Numbers may reflect the sensory intensity or the degree of liking and disliking. Interval scaling is best typified by the centigrade scale. Differences on the scale reflect differences in temperature. Ratios, however, are not meaningful (40° C and 20° C do not stand in a ratio of 2/1, although their difference is 20°). Just as differences in the centigrade scale can be maintained by adding a constant value (e.g., 100) to each number (which distorts the ratio, but leaves differences invariant), so can differences in the psychological interval scale be maintained, even with shifting zero points.

The Category Scale. The category scale is a variant of the traditional interval scale. The scale comprises a series of ordered categories, usually no more than 10 or 11. Observers are instructed to consider the categories as representing equal subjective intervals, so that the subjective difference between category 1 and category 2 is assumed (often by fiat) to equal the subjective difference between any other pair of adjacent categories (e.g., 7 and 8, or 3 and 4). Whether this is a reasonable assumption is open to question. Although the weight of evidence suggests that the category scale is not truly an equal interval scale,[17] the category scale is widely used, and many arithmetic and statistical operations are performed on the scale values, under the assumption that they display interval properties.

A number of things should be kept in mind when evaluating category scale data, especially if they relate to hedonic tone:

1. Sometimes the scale has a specific neutral point, and categories above that neutral point represent ordered degrees of liking, whereas categories below it represent ordered degrees of disliking. If the experimenter chooses not to designate a "neutral category" the observer himself may be presumed to implicitly designate a region on the scale as neutral, unbeknownst to the experimenter.[9]

2. Quite often the experimenter as-

signs descriptor words to each category value, to assist the observer in making judgments. Such consideration for the observer can produce an undesirable outcome in that the scale can then be used as a nominal one. The observer merely decides which descriptor best categorizes his feeling and the tasks become a nominal scaling or classification one, rather than a metric or measuring one.

3. Observers shy away from using the endpoints of the category scale, possibly because they are afraid of "running out of numbers." If a stimulus is given the highest category rating, an even more pleasing stimulus occurring later in the experiment would have to be assigned the same rating, even though the observer is well aware of a difference in pleasantness between the two. Therefore, the observer becomes conservative. (See ref. 17 for a discussion of this phenomenon, known as the category end effect.)

The 9 point hedonic scale shown in Table 20–I is a category scale, developed by Peryam and Pilgrim for the U.S. Army, at the Quartermaster Corps, in Chicago, Illinois.[13] It has been used extensively, for the evaluation of the sensory acceptability of foods to be procured by the military, and for measuring the likes and dislikes of young military men for several hundred food items.[7] The scaling operation is an efficient one, and the scale can be easily understood by the respondent. Although it is bias prone, the category scale, and especially the 9 Point Hedonic Scale, has become the major instrument in use today to measure hedonic response to food items.

TABLE 20–I

NINE POINT HEDONIC SCALE OF PREFERENCE

1	Dislike Extremely
2	Dislike Very Much
3	Dislike Moderately
4	Dislike Slightly
5	Neither Like nor Dislike (Neutral)
6	Like Slightly
7	Like Moderately
8	Like Very Much
9	Like Extremely

Figure 20–2 presents category scaling results for the taste intensity (darkened markers), and taste pleasantness (open markers) for various concentrations of glucose, sodium chloride, citric acid and quinine sulfate.[11] The observers in this experiment were 16 medical students at the St. John's Medical College, in Bangalore, India. Each observer made three category scale judgments of taste intensity for every stimulus (0 = no taste, 7 = extremely strong taste), and three category scale judgments of taste pleasantness or unpleasantness (1 = dislike extremely, 2 = dislike moderately, 3 = dislike slightly, 4 = like slightly, 5 = like moderately, 6 = like extremely, no designated neutral point).

The important points to note about these data (which allow for comparison of hedonic responses within the same individual, across concentrations) are:

1. The observers' numerical estimates of taste intensity grows in magnitude with stimulus concentration. The functions for different stimuli are, by and large, parallel with each other, except for perturbations at the lower end of the scale. This result differs from magnitude estimation of taste intensity (a ratio scaling procedure, discussed below), in that the growth rate of taste intensity with

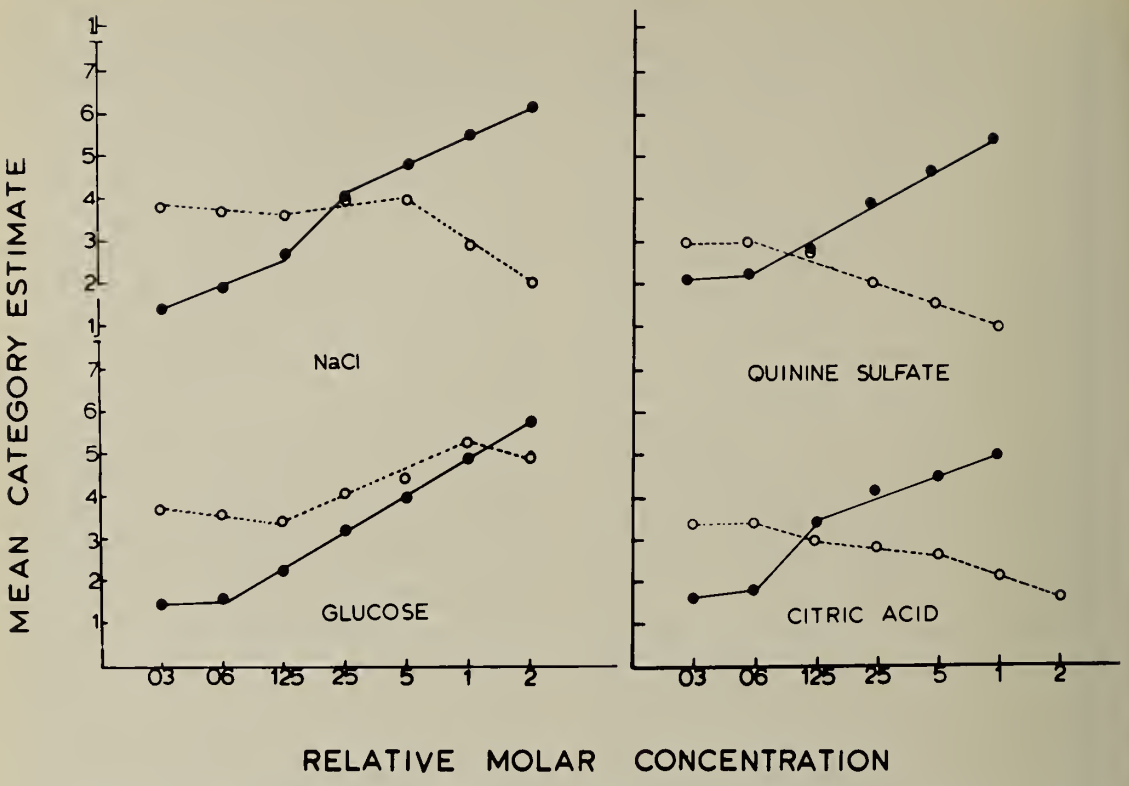


Figure 20–2. Relation between relative molar concentration, and both taste intensity (closed circles) and taste pleasantness (open circles). The data were collected using a 1–6 category scale for pleasantness and a 1–7 category scale for taste intensity. The data are from Moskowitz, Kumraiah, Sharma, Jacobs and Sharma (1975).

concentration varies from taste compound to taste compound.

2. The functional relations developed permit interpolations and, to a lesser degree extrapolations. Thus, the taste intensity (and taste pleasantness) of stimuli not directly tested can be estimated. In addition, the parameters of the taste-concentration function themselves may be compared across body states, satiety states, etc.

3. Analysis of the functional relation between judged taste intensity, judged hedonic level, and concentration, reveals that the observer responds to concentration shifts differently, when judging taste intensity and taste pleasantness. That is, the intensive and the hedonic attributes of the taste stimuli are functionally differentiated. The data suggest that whereas a unit increase in the perceived sourness of citric acid or the perceived bitterness of quinine sulfate produces a unit change in pleasantness, the relation of increases in perceived sweetness or perceived saltiness to pleasantness changes with intensity. With an appropriate scale inversion to reflect the fact that increasing the concentration of the sour and bitter stimuli produces decreases in pleasantness, the plots of taste intensity and hedonic tone as a function of

concentration are parallel to each other. No such parallelism occurs for sweetness and saltiness.

4. The pleasantness function for glucose sweetness exhibits a break (viz. a maximum point) at 1.0 M. This concentration is as sweet as 9% (W/V) sucrose. The same concentration (1.0 M glucose) has been shown to be maximally pleasant in other studies [8] in which the observers rated both the sweetness and the pleasantness of several dozen sugars in aqueous solutions.

Individual Differences in Hedonic Tone. Because category scaling permits assessment of the quantitative aspects of hedonic processes, experimenters can inquire into the generality of hedonic functions, such as those shown in Figure 20–2 (e.g., ref. 12). Are the pleasantness functions for sweetness and saltiness merely artifacts obtained by averaging together hedonic ratings from different individuals? Some of the individuals might find the sweet taste of glucose to be increasingly pleasant along the entire range of concentrations, in which case their judgments of intensity and hedonic tone would be positively correlated and their pleasantness functions might even be parallel to their own sweetness functions, when both are plotted against concentration. Some individuals might find just the opposite—that the sweet taste of glucose becomes increasingly unpleasant at higher concentrations. For these observers the pleasantness and sweetness judgments would be negatively correlated and the slope of their pleasantness functions would thus be opposite to that of their sweetness functions. Finally, there might exist another subset of individuals who find pleasantness to increase with sweetness up to a certain concentration, but then to diminish with further increases in sweetness. The concentration (or subjective sweetness level) corresponding to maximal pleasantness might itself be a random variable for this group. Depending upon the distribution of individuals in the population the pleasantness function for the sweet taste of glucose might appear as it does in Figure 20–2, or else might assume either an entirely ascending or an entirely descending function.

Figure 20–3 shows a set of pleasantness functions for both sucrose and sodium chloride that Pangborn obtained by category scaling.[12] According to Pangborn, the observers can be initially separated into three distinct clusters of respondents. One cluster indicates that they like increasing concentrations of sucrose, another cluster indicates that they dislike increasing concentrations of sucrose, whereas the third cluster indicates that they maximally prefer an intermediate concentration (3% W/V sucrose). Unfortunately, this data does not extend to higher concentrations, up to 9% and higher, where previous reports suggest that pleasantness might be maximal.

Ratio scales, power functions, and growth rates

Ratio scaling is the highest form of sensory measurement, and provides the experimenter with numbers that can be subjected to a variety of statistical operations. In the typical procedure, observers assign numbers to stimuli so that the ratios of these numbers reflect ratios of sensory

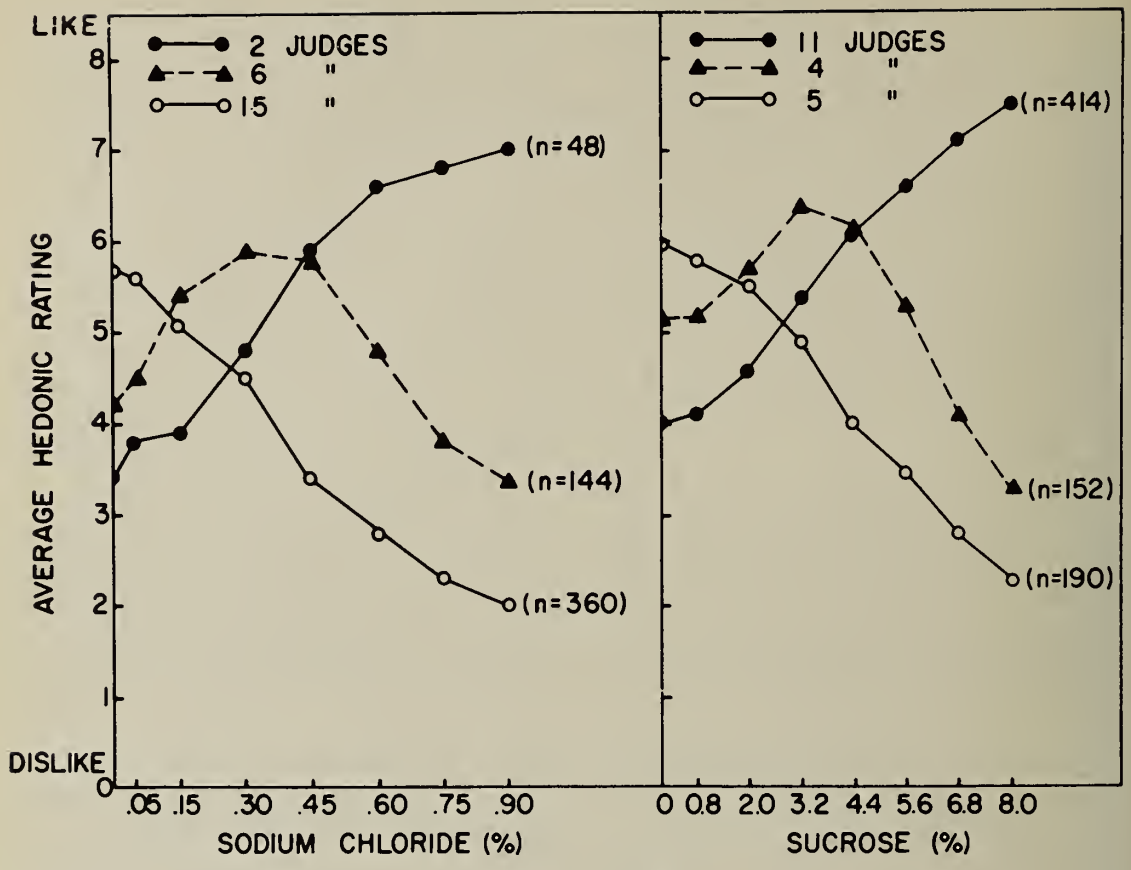


Figure 20–3. Relation between percent concentration and hedonic ratings of sodium chloride and sucrose. Note that the three different patterns suggest that individuals respond differently to the taste pleasantness or unpleasantness of these two, commonly used, sapid chemicals (Data from Pangborn, 1970 with the permission of the author and of Psychonomic Press, Inc.)

magnitudes (e.g., sweetness, bitterness, odor intensity) or of liking. Whether the numbers which the observer assigns truly display ratio-scale properties is an unresolved question. However, a variety of procedures have been developed which illustrate that the operation of number matching is consistent with the premises of ratio scaling (e.g., cross-modality matching, as described by Stevens [16]). The numerical assignment procedure is known as *magnitude estimation,* and has been used extensively to generate numerical relations between subjective estimates of perceived intensity (e.g., loudness, brightness, taste intensity) and instrumental measures of stimulus magnitude (e.g., acoustic energy, luminous energy, chemical concentration, respectively).

If linear coordinates are used to plot the relation of the numbers generated by magnitude estimation to the physical intensity of the stimuli, the functions may appear concave upwards, concave downwards or linear. However, in log-log coordinates the functions become linear, suggesting that sensory magnitude (S) can be re-

lated, in an *ad hoc* way, to physical intensity (I) by means of a power function: $S = KI^n$. The exponent n varies from one continuum to another. Functions that are concave upwards (accelerating functions) have exponents greater than 1.0. Good examples are the sweetness of glucose, the saltiness of sodium chloride, the perceived shock generated by current traversing the fingers, and the saturation of colors. Those functions which exhibit downward concavity include the perceived intensity for many different odorants, the sourness of acids and the bitterness of quinine and its derivatives. Saccharin sweetness also conforms to a decelerating function of concentration, in contrast to the sweetness function for glucose (and other sugars).

Although the metric properties obtained from magnitude estimation are more rigorous than those obtained from category scaling, nonetheless the general shape of the intensity functions are similar and the shape of the sweetness-pleasantness function is virtually identical. The pleasantness of sugar (and of sweetened foods, such as cherry flavored beverage, pudding, cake) first increases with concentration, reaches a maximum level (at the sweetness equal to that imparted by 1.0 M glucose) and then diminishes with increasing sugar level.[8,10]

The form of the pleasantness function (against concentration) can be more adequately explored with the method of magnitude estimation. As a first approximation, the increasing portion of the pleasantness function is governed by a slope (in log-log coordinates) which is lower than the corresponding slope (in that concentration region) governing the increase in perceived sweetness. That is, a 10-fold increase in concentration for levels lower than 1.0 M glucose may produce a 20:1 increase in perceived sweetness, but only a 3–4:1 increase in judged pleasantness. Although they are correlated, in that region, the growth of sweetness is more rapid than the growth of pleasantness.[8] As the concentration of glucose increased beyond 1.0 M the function for pleasantness turns over and pleasantness rating diminishes, usually at a rate which is the inverse of that for concentration (viz. a 2:1 increase in concentration produces a loss of about 50% in pleasantness). Relatively little data is available, however, on the parameters of the descending portion of the pleasantness function for sucrose, whether by magnitude estimation or by category scaling.

LINGUISTIC ASPECTS OF HEDONIC MEASUREMENT

What does the concept "pleasantness" or "unpleasantness" mean to the individual? That question may seem simple, although Beebe-Center's historical review of research on pleasantness or unpleasantness [1] suggests that what is eminently clear to the intuition is, more often than not, unclear to the intellect. Sensory magnitude may be correlated with increases in physical intensity, and the experimenter can objectively show the observer such correlations. The experimenter need only increase the electrical energy applied to earphones, and show the observer the

readings on a voltmeter, while the observer attends to increases in subjective loudness. On the other hand, pleasantness and unpleasantness do not necessarily correspond to an easily manipulated physical continuum and thus may be viewed as lying solely within the individual. Biological or maturational changes in the observer may produce changes in apparent pleasantness (cf. Engen [5]). In addition, a stimulus which seems pleasant in one circumstance may be repugnant under another (e.g., the odor of cabbage at the dinner table vs. the same odor in a perfume shop).

If situational factors are so potent an influence on hedonic judgments, and if an experimenter's verbal instructions to judge pleasantness rather than sensory intensity can provoke an entirely different subjective function (for estimates plotted against physical magnitude), then at least one serious problem arises. How is the experimenter to decide what the organism attends to when making a judgment and exhibiting behavior? Much of the work that has been reported on animal choice behavior is open to the question of what aspect, sensory or hedonic, controls or determines the behavior. Behavioral measures alone may not necessarily distinguish between pleasantness and sensory magnitude. Choice behavior and ingestive behavior, which seem so valid as indications of preference, break down if the experimenter tries to determine which attribute, taste intensity or taste preference, is responsible for the behavior. Only the presence of language, and the ability to verbally state the difference between liking and sensory intensity, can allow the experimenter to distinguish between the two possible determinants of behavior. Retrospective views of behavior patterns do not, by themselves, indicate which attribute was operative at the time of the behavior.

A human observer making taste judgments may be instructed to attend to either sensory intensity or to pleasantness. In at least one situation ratings of the sweetness intensity for a variety of foods could be distinguished, functionally, from judgments of acceptability by this method.[10] The observer generates a different appearing *pattern* for sweetness (vs. concentration) than for pleasantness (vs. concentration). These two patterns can only be contrasted with each other if they are elicited from the same observer, under conditions that differ only with respect to verbal cues that both the experimenter and the observer understand properly.

AN INTERPRETATION THEORY OF HEDONIC JUDGMENTS

The foregoing sections set forth the premise that much of our response to external stimuli (at least in terms of psychophysical scaling) is cued by verbal labels, such as intensity and pleasantness. To what degree is the hedonic response to a stimulus the result of interpretation? If observers receive and operate only upon sensory information, and yet generate reproducible and different functions for intensity and pleasantness, one is lead to ask to what degree the pleasantness function is a product of

secondary processing of the sensory information?

There are at least two possibiities to explore. One possibility is that, while the organism has available to it a sensory system which faithfully mirrors physical intensity by one or another transform, it deals with hedonic information categorically. Hedonic tone would have no true variation in magnitude, although sensory intensity would. Possibility 1 implies that the hedonic functions obtained when the observer estimates taste pleasantness depend upon a misunderstanding of the observer's instructions. Variations in pleasantness judgments with physical intensity might occur because the observer, incapable of distinguishing variations in hedonic tone (or even realizing that hedonic tone has no intrinsic variation), attends to variations in sensory magnitude. The ratings which the observer gives are reflections of a judgment of sensory magnitude coupled with the presence or absence of a constant, fixed, underlying hedonic tone. For acids and bitters there may be no problem in rating, because increasing concentrations of acid never become pleasant, nor do increasing levels of bitters. On the other hand, sometimes too-sweet products change from pleasant to unpleasant. If this shift is a random variable, then we might expect to see an attenuated intensity function, which occurs for sweetness and for saltiness.

Possibility 2 posits that there are true hedonic functions for some continua, but not for others. I have labelled these continua Type I and Type II, respectively.[9] Type I continua are those whose hedonic function parallel the intensity function, and the experimenter has no a priori way to demonstrate whether the observer is actually estimating sensory intensity or hedonic tone. Among Type I continua are: the annoyance of noise; the pleasure of touch; the unpleasantness of sourness; the unpleasantness of butanol odor. Type II continua fail to show functionally equivalent sensory intensity and hedonic functions. They include the pleasantness of sugar, the pleasantness and unpleasantness of sodium chloride, and the pleasantness of some odorants.

Obesity and hedonics

In some experiments in which obese observers gave ratings for the pleasantness of sucrose and sodium chloride, increasing concentrations of sucrose occasion a monotonic decrease (or increase) in pleasantness in contrast to the inverted U shaped function which normal weight individuals exhibit.[6] This monotonicity suggests quite strongly that obese subjects do not really attend to the hedonic tone of the sweet taste. Rather, they appear to attend primarily to the sweetness of the sucrose solution, and misconstrue that sweetness to be pleasantness. In terms of the interpretation theory proffered above, obese individuals do not have access to an underlying pleasantness continuum, whether this continuum be simply a global, all-or-none feeling about pleasantness (viz. possibility 1) or else a true hedonic continuum operating in conjunction with the sensory intensity continuum (viz. possibility 2, Type II continua). Rodin *et. al.* [14] found similar results with the pleasantness ratings of sweetened cherry flavored beverages. Obese girls in a summer camp, on a weight reduc-

ing regimen, found the taste of the beverage to increase in pleasantness monotonically with concentration. Girls of more normal weight showed the expected breakpoint in the pleasantness function. This weight dependent failure to display the breakpoint implies that the obese lack the ability to distinguish between pleasantness and taste intensity.

Alliesthesia and hedonics

Cabanac [2] has proposed that the internal physiological state may alter our perception of the pleasantness (or unpleasantness) of external stimuli. He has reported that after the ingestion of a satiating load of glucose observers find taste to be less pleasant than when they were hungry. This internal regulation of hedonic responsiveness is called alliesthesia, and operates on a variety of sensory systems in which hedonic tone enters as a constituent of the sensory experience (e.g., taste, smell, temperature).

Cabanac's alliesthesic hypothesis may be a specific case of the more general hedonic process outlined here. Satiated individuals may lose the ability to distinguish the hedonic tone of stimuli which impinge upon them, and rely simply upon the sensory information. For sweetness that loss would change the taste hedonic from Type II (where intensity and pleasantness are not equivalent to each other) to Type I (where intensity and pleasantness are equivalent to each other). Such a shift would occur in two seemingly different ways.

1. The pleasantness function for the sweet taste would fail to show a breakpoint, although pleasantness would still rise with concentration. After satiation, 1.0 M would no longer be a critical glucose level, since the observer no longer has graded hedonic information available to him. Type I behavior would prevail, and the pleasantness function would approximate the sweetness function. On the other hand, no such dramatic shift need occur. The hedonic function which underlies sweetness perception might be diminished in importance, so that the observer attends only somewhat to the hedonic tone of the taste, and is no longer able to home in on the critical sensory level of sweetness above which the sweet taste fails to increase in pleasantness with concentration (viz. 1.0 M glucose). Just such an extraordinarily attenuated, but not eliminated, hedonic function was found by Moskowitz, Kumraiah, Sharma, Jacobs and Sharma [11] for Indian medical students who were artificially sated with a glucose load (1.5 g/kilogram body weight). Whereas the unsatiated pleasantness function for glucose taste for equivalent groups (tested under the same regimen) who showed a breakpoint at 1.0 M glucose, the pleasantness function for the satiated group failed to show the breakpoint.

2. The pleasantness function might invert, so that the observer would report that the taste previously pleasant is unpleasant. The unpleasantness rating would either be constant across concentrations (showing a global negative hedonic tone for the taste) or else the unpleasantness of the taste would increase in magnitude with concentration.

SPECULATIONS ABOUT STUDIES OF THE ONTOGENY OF TASTE PREFERENCES

At what stage can we assume that the infant becomes aware that sweetness is pleasant, whereas sourness, bitterness and high levels of saltiness are unpleasant. Can the infant differentiate between the sensory and the hedonic aspects of stimuli immediately, or is this learned during development? Considerable evidence points to the responsivity of infants (primarily neonates) to sweet solutions. The hedonic character of this response is always inferred, however, from the anthropomorphic conclusions of the investigator. Since the infant does not have a language (or its nuances are not yet sufficiently known to distinguish between aspects, such as sensory intensity and sensory pleasantness), our studies of infants must draw conclusions about behavior alone, without specifying which of two correlated aspects of the stimulus produce that behavior. The same difficulty pervades non-verbal measures of human adult behavior. Knowledge that one beverage was drunk in five times greater quantities than another beverage does not allow us to conclude that it was five times more pleasant rather than five times more sweet (unless we predicate the definition of pleasantness on the operation of drinking in a choice situation).

The foregoing train of logic is initially discouraging, for it implies that researchers in neonatal and infant behavior may be left without the ability to distinguish behavior based on judgments of sensory magnitude from judgments based on hedonic tone. Nonetheless, this problem opens up a fertile avenue of research involving the ontogenetic comparison of sensory and hedonic properties of the same taste materials. The observers would be human beings, during various phases of development, from infancy (without language) through childhood (where language develops) and on through adulthood (where language and meanings are refined). Concomitant with this study of responsiveness to external stimuli would be a study of the meaning, behaviorally or linguistically (or both), of pleasantness of sensory magnitude. It should be possible to trace the response of the human to a beverage backwards, starting from adulthood and moving into childhood and infancy, all the while simultaneously finding behaviors which differentiate between pleasantness and taste intensity. The tools of the experiment and the research paradigms will shift with the capacities of the observer. Adults and children with good vocabularies will be able to state the difference between their perception of the sweetness of the beverage and their perception of its pleasantness (how much they like it, how acceptable the beverage is—the set of distinctions itself suffices for an entirely different research project). Validations of the differentiation between sweetness and pleasantness might be obtained by testing those concentrations at which sweetness is extreme, but pleasantness (or intake) has dropped off. Paradigms which differentiate, operationally, between pleasantness and sweetness could maintain one response (e.g., drinking, magnitude estimation) when the cue is sweetness,

and maintain another response for the same stimulus (e.g., rejection, magnitude estimation) when the cue is pleasantness.

It may ultimately be possible to show that infants do perceive differences between the magnitude or intensity of a stimulus and its hedonic tone. Although markedly different functions for sweetness and pleasantness versus concentration can be demonstrated in adults the exact form of the analogous function in youngsters may be elusive.

REFERENCES

1. Beebe-Center, J.G.: *The Psychology of Pleasantness and Unpleasantness.* New York, Van Nostrant Inc., 1932.
2. Cabanac, M.: Physiological role of pleasure. *Science,* 173: 1103–1107, 1971.
3. Chappell, G.M.: Flavour assessment of sugar solutions. *Journal of the Science of Food and Agriculture,* 4:346–350, 1953.
4. Engel, R.: Experimentelle Untersuchungen uber die Abhangigkeit der Lust and Unlust von der Reizstark beim Geschmacksinn. Pfleuger's Archive fur die Gesamte Psychologie, 64:1–36, 1928.
5. Engen, T.: The potential usefulness of sensations of odor and taste in keeping children away from harmful substances. *Annals of the New York Academy of Sciences,* 237:224–228, 1974.
6. Grinker, J. and Hirsch, J.: Metabolic and behavioral correlates of obesity. In: *Physiology, Emotion and Psychosomatic Illness.* Amsterdam, Holland Elsevier, 1972, 349–374.
7. Meiselman, H.L., van Horne, W., Hasenzahl, B., and Wehrly, T.: The 1971 Fort Lewis Food Preference Survey. Technical Report 72–43, U. S. Army Natick Laboratories. Natick, Mass., 1972.
8. Moskowitz, H.R.: The sweetness and pleasantness of sugars. *American Journal of Psychology,* 84: 387–405, 1971.
9. Moskowitz, H.R.: Hedonics and intensity: Symmetries and asymetries. (In preparation)
10. Moskowitz, H.R., Kluter, R.A., Westerling, J., and Jacobs, H.L.: Sugar sweetness and pleasantness: Evidence for different psychological laws. *Science,* 184:583–585, 1974.
11. Moskowitz, H.R., Humaraith, V., Sharma, K.N., Jacobs, H.L., and Sharma, S.D.: Effects of hunger, satiety and glucose load upon taste intensity and taste hedonics. *Physiology and Behavior.* In press.
12. Pangborn, R.M.: Individual variations in affective responses to taste stimuli. *Psychonomic Science,* 21:125–126, 1970.
13. Peryam, D.R. and Pilgrim, F.J.: Hedonic scale method for measuring food preferences. *Food Technology,* 11: 9–14, 1957.
14. Rodin, J., Moskowitz, H.R., Fleming, B., and Bray, G.A.: Relationship between obesity, weight loss and taste responsiveness. *Physiology and Behavior.* In press.
15. Stevens, S.S.: On the theory of scales of measurement. *Science,* 103: 677–680, 1946.
16. Stevens, S.S.: The quantification of sensation. *Daedalus,* 88: 606–621, 1959.
17. Stevens, S.S. and Galanter, E.: Ratio scales and category scales for a dozen perceptual continua. *Journal of Experimental Psychology,* 54:377–411, 1957.
18. Thurstone, L.L.: A law of comparative judgement. *Psychological Review,* 34: 273–286, 1927.

Chapter 21

IMPLICATIONS OF RESPONSIVENESS TO SWEET TASTE FOR OBESITY

Judith Rodin

The association between taste responsiveness and overweight has been widely demonstrated in both animals and humans for a variety of experimentally and genetically produced obesities. Animals with ventromedial hypothalamic lesions typically show greater responsiveness to the sensory qualities of their food than normal weight intact animals and great increases in consumption can be obtained simply by adding dextrose to standard food.[5,35,38] The eating behavior of many varieties of genetically obese mice [1,7,33] and of hibernators during the phase of weight gain and obesity prior to hibernation [20] is also highly responsive to taste.

TASTE RESPONSIVENESS IN OVERWEIGHT HUMANS

Like the heavy animal, the obese person often reacts strongly to the taste of his food. Hashim and Van Itallie [11] gave clinically obese patients nothing to eat but a bland, unappetizing liquid formula for several weeks. The patients typically consumed only a few hundred calories per day of this food, whereas they had previously been eating several thousand calories of the standard hospital diet. Weight reduction during long periods of exclusive access to the formula diet was dramatic. In contrast, normal-weight subjects given the formula maintained normal caloric intake and experienced little or no weight loss.

It seems likely that the obese patients in Hashim and Van Itallie's experiment consumed very small amounts of formula simply because they did not like the taste. A direct test of this hypothesis was provided in an experiment by Nisbett.[21] Nisbett asked obese* and normal subjects, as well as under-weight subjects, to participate in an experiment examining "the effects of hunger on ability to concentrate." All subjects were told that they were in the experimental condition and would be fed prior to taking the concentration tests. The food which they were asked to eat was vanilla ice cream in one of two forms: either an excellent and expensive French vanilla or vanilla which had been adulterated with quinine. To avoid suspicion, the ice cream was de-

*Percent overweight in this and subsequent experiments reported here was determined on the basis of Metropolitan Life Insurance norms (Ref. 17) calculated for height and age. Heavy subjects were 15% or more overweight by these standards.

Acknowledgment: The author's research discussed in this paper was supported by NSF Grant GS–37953.

scribed to all subjects as having a new "vanilla bitters" flavor. The three weight groups responded in dramatically different ways to the taste manipulation. Overweight subjects were the most responsive to the taste, eating far more of the good-tasting than of the adulterated ice cream. Underweight subjects were far less responsive, eating more nearly equivalent amounts of good and bad tasting ice cream. Normal subjects displayed an intermediate responsiveness to taste.

Subjects were also asked how much they liked the ice cream they had been given. These ratings provide for more informative analyses than the two-level, good-bad manipulation. Figure 21–1 presents the amount of ice cream eaten as a function of subject's weight and ratings of the ice cream. Underweight subjects ate more disliked ice cream than did normal subjects and less liked ice cream. Overweight subjects ate roughly the same amount of disliked ice cream as other subjects, but far more liked ice cream.

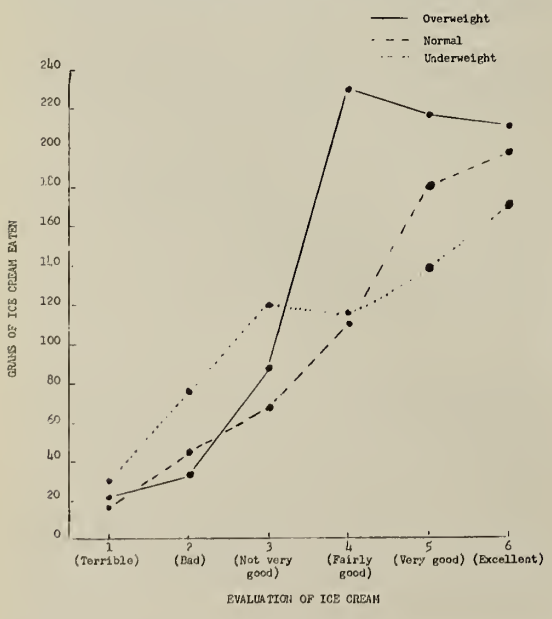


Figure 21–1. Grams of ice cream eaten as a function of ratings of its taste.

In a second study, Nisbett, in collaboration with Harry Jacobs and Joel Sidel [22] examined the response of volunteer taste testers to the taste of cake samples which ranged in quality from the best available in the Army to unpleasant experimental preparations such as a cake stored at 100° for 100 days. The height and weight of taste testers were recorded. Cake samples were weighed prior to and after sampling. Figure 21–2 presents the amount of cake eaten as a function of the weight of the volunteer and his evaluation of the cake sample. The pattern is similar to that of Figure 21–1. Underweight subjects ate more disliked cake and less extremely liked cake than normal subjects. Overweight subjects ate roughly the same amount of disliked cake as other subjects but considerably more of the cake which they liked.

Using a different paradigm, Cabanac suggested that overweight and normal weight people are most dissimilar in their response to sweet

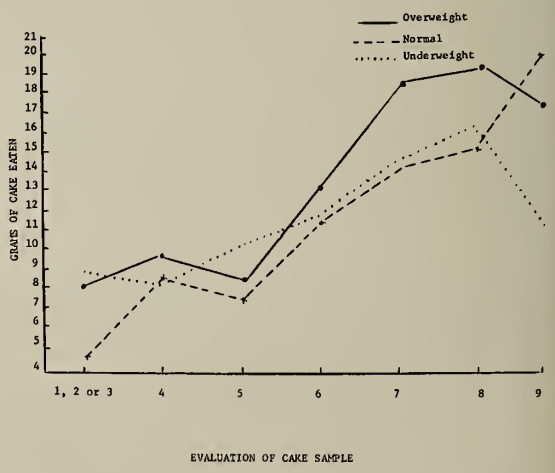


Figure 21–2. Grams of cake eaten as a function of subject's weight and evaluation of cake sample.

taste after a glucose preload.[3] While hedonic ratings of oral sucrose solutions varying in sweetness were high for both obese and normal patients after a 12 hour fast, the ingestion of 50 grams of glucose in 200 ml. of aqueous solution produced a dramatic drop in affective response to sucrose for normal weight individuals. The obese, on the other hand, appeared unaffected by the glucose gastric load and continued to give high positive ratings to the stimuli.

CORRELATION AND CAUSE

Experiments which demonstrate a strong association between obesity and sweet preference, naturally lead to speculation about the etiologic significance of responsiveness to sweet taste for the development of obesity. As many of us have considered this issue [22,23,34] there seem three good possibilities:

1) Heightened taste responsiveness may be one of the contributing factors to overeating and obesity. This hypothesis suggests that there are individuals who, either biologically or through early experience, are highly responsive to palatable foodstuffs and it is this condition which contributes to overeating and consequent weight gain.
2) The obese condition itself may alter taste responsiveness such that once an individual attains a certain degree of overweight, his responsiveness to good tasting food is enhanced.
3) The two are correlated and can co-occur, but may both be caused by a third factor. This hypothesis suggests that there is no direct causal relationship between taste responsiveness and obesity.

Unfortunately, or perhaps interestingly, there is some evidence for all three points of view in both the animal and human obesity literature.

Evidence with ventromedial-lesioned animals

The ventromedial hypothalamic (VMH) animal will serve as an example since it is the preparation I am most familiar with. The first hypothesis is supported by data which suggest that these animals attain their highest degree of obesity only when given good tasting food which is either sweet-tasting or high in fat.[18,35] This diet typically produces a dynamic bout of overeating and weight gain even among VMH animals in the static phase of obesity.[5] The second alternative is supported by Teitelbaum's [38] work that indicates that only animals which have already become quite obese actually show hyperresponsiveness to sweet taste. Animals in the pre-obese, dynamic stage are only slightly more finicky than control animals. The third alternative is supported by Graff and Stellar's [8] finding that the placement of the lesion itself determines whether obesity and differential taste responsiveness co-occur or are produced independently.

Evidence with human obesity

Support for the presumption that taste responsiveness precedes and contributes to the development of obesity comes primarily from the

demonstration that currently normal weight individuals who were once overweight, are still highly responsive to taste and ingest large quantities of preferred food.[21,23] However, some physiological changes associated with obesity remain even after weight loss. In juvenile onset obesity, for example, the number of fat cells remains elevated regardless of weight lost.[14] Nonetheless, recent work has shown that there are some highly taste responsive normal weight individuals who have never been overweight.[12] These people are, by self report, restrained eaters who consciously and continuously monitor their eating. They are also highly responsive to external cues such as fear-provoking stimuli.[13] Their behavior lends support to the assertion that responsiveness to sweet taste as well as other potent external cues, may contribute to the development of obesity in the absence of severe monitoring.

Let us now consider the second alternative, tested by attempts to demonstrate that overweight causes differential sweet preference. This question has been approached by considering whether individuals who are obese at birth show differences in taste responsiveness. Nisbett and Gurwitz [24] reported that heavy infants drank more glucose enriched sweet tasting formula than lighter infants when fed ad lib. Yet, Desor, Maller and Turner [6] recently pointed out that the finding that heavy babies prefer sweet solutions in their 3 minute-taste tests depends on how weight categories are defined. Furthermore, overweight is more critical to a test of the causal hypothesis than heaviness, and Nisbett and Gurwitz actually found no relationship between their index of overweight, "weight/length,[3]" and responsiveness to sweet taste, thus weakening the evidence for this alternative.

Finally, there is less direct evidence with humans for the third alternative—that is that weight and taste responsiveness are merely correlated and not causally linked. It may be however, that people biologically predisposed to be fat may also have a predisposition, at the level of the CNS, to be highly taste responsive.[22]

Thus, neither the direction nor the very existence of a causal relationship between obesity and taste responsiveness has been well established. Before further attempts are made to deal with the etiological significance of sweet taste responsiveness for obesity, we should define more clearly the components of taste to which obese subjects differentially react and precisely how this can influence food intake.

Relationship between weight and sensory aspects of sweet taste

Does the difference between the weight groups lie in their judgment of the presence and intensity of sweetness? Grinker and her coworkers [9,10] have demonstrated that the difference does not seem to be one of discrimination or detectability. Do the groups differ then in how pleasant they find sweet-tasting substances. Grinker's examination of this question showed that at higher concentrations of sucrose, overweight and obese subjects actually rated the sweetened liquid less rather than more pleasant than normals and drank less. These experiments all used sucrose and water solutions as the test stimuli and

examination of the data reveals that the mean ratings for all weight groups were generally in the neutral range of hedonic value. Perhaps, the stimuli were simply not sufficiently palatable to elicit differences as a function of weight. Since preference is determined by both hedonic processes and dietary habits,[42] we decided to repeat these experiments using a familiar and highly palatable taste substance.[28] The experiment examined whether the obese are more sensitive to, and prefer increases in, the sweetness of a rich and good tasting substance and whether preference leads to instrumental behavior to obtain the substance.

Judgments

To a standard, pre-sweetened milkshake, sucrose was added to produce sugar solutions equal to .17, .34, .51 and .68 molar solution in the shake. These four and the unadulterated milkshake were presented in 5 ml. cups to each subject, in random order. The subject took a sip of each, spit it out, rated it on a 9 point scale for intensity of sweetness and rinsed his mouth with water.[19] This procedure was repeated for taste pleasantness ratings.

As Table 21–I shows, there were no significant weight group differences in intensity or pleasantness ratings for the five increasingly sweet substances even when the food was tasty and familiar. However, the weight groups were dissimilar in the degree of their reaction. A simple effects interaction test indicates that the difference between the mean hedonic ratings for the most and least preferred taste substance was significantly greater for overweight subjects than for normals ($F_{1,72} = 4.01, p < .05$).

Motivational effects

To assess motivation to obtain sweet taste, subjects next were promised a full glass of milkshake if they solved a spatial puzzle. They were instructed to work on the puzzles only as long as they wished since this part of the experiment was simply a pretest of several puzzles for a later study. The puzzles were actually unsolvable. For one third of each weight group, every subject was offered his or her most

TABLE 21–I

MEAN INTENSITY AND PLEASANTNESS RATINGS FOR MILKSHAKES VARYING IN SUCROSE CONCENTRATION

GROUP	*CONCENTRATIONS*				
	Base	*.17M*	*.34M*	*.51M*	*.68M*
	Intensity of Sweet Taste				
Overweight	5.66	6.27	7.13	8.47	8.97
Normal-weight	5.73	6.63	7.34	7.83	8.14
	Pleasantness of Sweet Taste				
Overweight	7.57	7.59	8.37	7.01	7.46
Normal-weight	7.93	8.03	8.02	7.56	7.13

Note: On the intensity scale 1 = no sweet taste, 9 = extremely strong sweet taste.
On the pleasantness scale 1 = extremely unpleasant, 9 = extremely pleasant.

TABLE 21–II

MINUTES SPENT WORKING ON THE UNSOLVABLE PUZZLE FOR PREFERRED AND UNPREFERRED REWARD

REWARDS	*SUBJECTS*	
	Overweight	*Normal weight*
Least preferred milkshake	2.70	2.92
Most preferred milkshake	7.96	5.02
Most preferred milkshake after preload with same milkshake	9.37 [a]	3.64 [a]

Note: After 10 minutes, any subject still working on the puzzle was stopped and given a score of 10.
[a] $p<.01$

preferred milkshake for solving the puzzle and another third was promised their least preferred. The remaining third in each weight group was also offered their most preferred milkshake for solving the puzzle and given an 8 ounce glass of that milkshake 20 minutes before they began working on the puzzle.* As shown in Table 21–II overweight subjects worked longer, on the average, than normals for their most preferred milkshake and spent about the same time working for the least preferred substance. Most interesting, the preload apparently increased the overweight subjects' motivation to obtain more milkshake whereas in normal weight subjects it had precisely the opposite effect, rendering their persistence comparable to that demonstrated for the least preferred substance.

The data suggest that preference does affect motivation, and that it does so more strongly for overweight individuals. Ingesting the highly preferred substance enhanced the motivation of this group. But normals appear to respond to satiety cues that decrease their motivation to obtain more. For the obese it appears that short term satiety cues are either not working properly or are overridden by affective responsiveness to the good tasting milkshake.

*If the subjects had not stopped working on the puzzle after 10 minutes, they were stopped and assigned a score of 10

Influence of perceived pleasantness on food intake in the obese

The finding that sensitivity and preference differences between weight groups are not evident when the intensity of the taste substance is varied, suggests at least two possible explanations. First, it may be that a different aspect of taste influences reactivity in the obese, for example, texture, temperature, or the ease of getting the taste. Second, it may be that so-called differential taste responsiveness is actually a difference in food intake alone and not a difference in either discrimination or preference. The next experiment was designed to test whether the effort involved in obtaining the substance would influence food preference and ingestion in overweight persons, and to explore alternative definitions of overweight.

Overweight and weight stability

We considered how perceived pleasantness influences ingestion and

whether differences between weight groups could be attributed to differences in set point for adipose tissue mass.[28] Cabanac and his colleagues [2,3,4] have suggested that if an organism is below its set point, internal physiological cues signalling short term changes in nutritional state are ignored. While a glucose load in an undeprived organism renders sweet solutions unpleasant,[4] the same load during severe long-term deprivation has no effect on the rated pleasantness of the sweet solutions. Cabanac et al attribute this result to the fact that the adipose tissue mass in the deprived organism is below its biologically determined set point, thus heightening the attractiveness of sweet taste by rendering satiety mechanisms ineffectual. Nisbett [23] has suggested that most moderately overweight individuals are probably below set point if they diet in response to social pressure and consequently also fit this description of a deprived organism. In a reanalysis of some earlier data, Nisbett classified overweight subjects as below set point if they were less than 40% overweight. Individuals who ignored social pressure and became extremely overweight were presumed to be at or above set point and consequently were not expected to show the energy deficit pattern. Normal weight subjects were considered below set point if they had once been overweight. Nisbett found that subjects in weight groups classified as below set point were, on the average, more responsive to good and bad tasting ice cream than subjects designated at set point.

While the notion of a biologically determined set point is intuitively appealing and theoretically potent, Nisbett's operational definition for humans seems open to question. There appears to be no *a priori* reason to assume that one who is 40% overweight is actually closer to his own biological set point than one who is 20% overweight, that is, if degree of appropriate adipose tissue mass is continuously and normally distributed in the population. Indeed it is possible that the 40% individual is above his set point, a state whose physiological implications are unspecified. These considerations suggest that degree of overweight may not be the best way to view set point differences. Instead, closeness to set point might be inferred from long term weight stability. For the present study we assumed that individuals who reported no dietary restrictions and whose weight showed little yearly fluctuation were closer to their biological set point than others whose weights were very unstable.

An operational definition of nearness to set point, more stringent than those that had been previously applied to humans, was based on three weight measures taken over the course of approximately one year. The first weight was obtained during the spring semester of the school year when medical forms for the incoming freshman class were returned to the admissions office. The following fall, under the guise of a health service weight survey, 300 potential subjects were weighed and a weight history questionnaire was administered which was designed to determine the degree of weight fluctuation in the past few years, childhood weight history and eating practices, and current dietary habits. A third weight measure was taken the following spring when the

experiment was actually conducted.

On the basis of these self-report and measurement data, subjects were considered below set point if a) their weights had fluctuated more than 7% in the past 2 years, and b) they reported usually not eating whatever and as much as they wished. Subjects assumed to be operating at or close to set point were those a) whose weight had not fluctuated more than 3% during the last 2 years, b) who did not restrict their eating, and c) who weighed as much as they had ever weighed.

Subjects in these two groups were further divided into three subgroups according to degree of overweight. These groups were 1) 60% or more overweight (obese), 2) between 15% and 40% overweight (overweight), and 3) between −10% and +10% overweight (normal-weight). There were 10 subjects in each of the six groups.

Hedonic judgments and intake

Forty-five minutes after a uniform preload of a sandwich and a glass of water, subjects were allowed unlimited access to chocolate milkshake as part of a taste test and were instructed to drink as much milkshake as needed in order to carefully complete a lengthy taste questionnaire. All subjects were given 36 ounces of chocolate milkshake in a 48 ounce opaque plastic container to reduce visual food-relevant cues.

There was large within group variability in pleasantness ratings for all weight groups and consequently we divided subjects into those who rated the milkshake as neutral or less and those who rated it as moderately pleasant or above (Table 21–III). Of those subjects who rated the milkshake as pleasant, overweight subjects found it significantly better and liked it significantly more than either obese or normal weight subjects ($F=5.89$, $df=1/48$, $p<.025$). Within weight groups, weight stability or unstability had no significant effect on taste ratings.

Similarly, the milkshake intake data shown in Figure 21–3, reveal no effect of set point as defined here. Degree of overweight however did have a significant effect ($F = 6.53$, $df = 2/54$, $p<.01$). Specific comparisons indicate

TABLE 21–III

INTENSITY OF HEDONIC RESPONSE BY SUBJECTS AT AND BELOW SET POINT AND DIFFERING IN DEGREE OF OVERWEIGHT (WIDE STRAW CONDITION)

	By Current Weight		
By History	*Normal weight*	*Overweight*	*Obese*
	Liked Milkshake (rating + 1 to + 3)		
At set point	1.67 (4) *	2.80 (5)	1.60 (5)
Below set point	1.67 (3)	2.40 (5)	1.40 (5)
	Disliked Milkshake (rating −3 to 0)		
At set point	−1.17 (6)	−2.20 (5)	−1.40 (5)
Below set point	−1.43 (7)	−1.20 (5)	−1.40 (5)

*Bracketed number represents the number of subjects in each category.

that overweight subjects as a group consumed significantly more than did either obese ($F = 5.04, df = 1/54, p < .05$) or normal weight subjects ($F = 8.84, df = 1/54, p < .01$). The overall correlation between preference and intake was highly significant suggesting that the more subjects liked the milkshake the more they drank ($r = .41, p < .01$). This was especially true for overweight ($r = .51, p < .01$) and obese ($r = .58, p < .01$) subjects. Several normals who liked the milkshake however did not drink greater amounts ($r = .06$), presumably because they were responding to satiety cues from the sandwich preload. These results are consistent with earlier findings that overweight subjects consume more of highly preferred but somewhat less of nonpreferred foods in comparison with normals.[21,25]

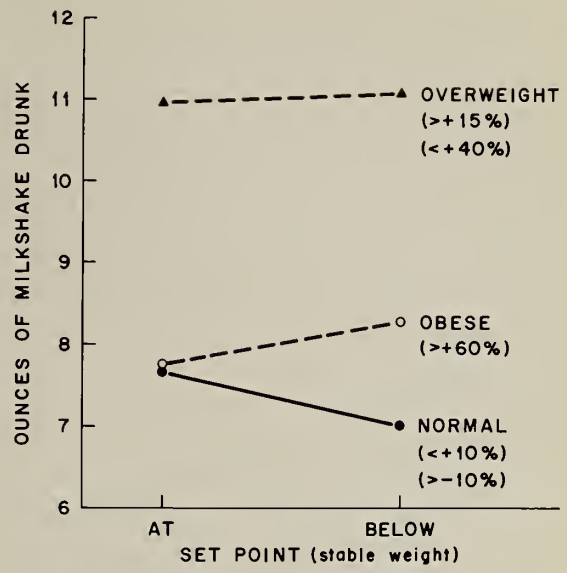


Figure 21–3. Mean ounces of milkshake drunk in easy straw condition by set point and below set point subjects differing in degree of overweight.

Effort

If overweight individuals like better and consume more of a highly preferred milkshake, it might be expected that they would be more willing than normals to work to obtain this food. The experiment described above was actually a repeated measures design in which there were two taste rating sessions, two to three weeks apart. In each session, half the subjects in each weight group tasted the milkshake through a thin tube with an inside diameter of .106 inches making it quite difficult to drink the thick milkshake. The other half drank through a wide tube with an inside diameter of .157 inches. Figure 21–4 shows the difference in amount consumed through the difficult and easy straws.

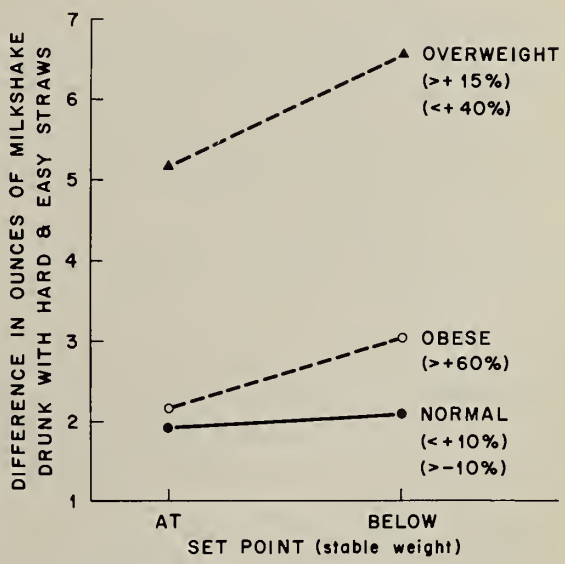


Figure 21–4. Mean difference in ounces of milkshake drunk between hard and easy straw conditions in set point and below set point subjects differing in degree of overweight.

It is clear that overweight subjects were most deterred by the effort involved in sipping through the very

thin straw. Paired comparison tests show that overweight subjects were significantly more deterred by the difficult straw than were either normal weight ($F = 9.01$, $df = 1/54$, $p < .01$) or obese subjects ($F = 7.32$, $df = 1/54$, $p < .01$). There was also a significant main effect for weight stability ($F = 4.59$, $df = 1/54$, $p < .05$) but no interaction effects.

Earlier we demonstrated that overweight subjects will work longer at puzzles to obtain a preferred milkshake. Before concluding that if too much effort is involved, it does not matter how much they like the taste, let us examine the taste ratings given to the milkshake when the difficult straw was used. All subjects actually found the milkshake *less* pleasant when they had to work hard for it. The effect was greatest for overweight subjects particularly when, as indicated in Table 21–IV, the groups were again divided into those giving above and below neutral ratings. Overweight subjects gave taste ratings which were significantly less positive (t diff = 2.33, $df = 8$, $p < .05$) and somewhat more negative (t diff = 2.17, $df = 10$, $p < .10$) when using the narrow straw than when using the wide straw.

It should be remembered that this was a repeated measures design with counter-balanced order of presentation so these were the same subjects who rated the milkshake as more pleasant when they used the easy straw. Subjects were not informed that they were rating the same milkshake in both situations. These results, combined with those from the "easy" straw condition, suggest that what overweight subjects like, they seem to like more strongly than other weight groups, and what they dislike, they dislike more strongly. This finding supports other demonstrations that overweight people are more responsive to salient cues,[34] if we assume that highly positive or negative foods are potent stimuli. Overweight subjects also appear more likely to determine preference for a taste substance on the basis of the ease or difficulty of getting it into the mouth and degree of liking, in turn, significantly influences ingestion.

The data replicate Nisbett's [23] find-

TABLE 21–IV

INTENSITY OF HEDONIC RESPONSE BY SUBJECTS AT AND BELOW SET POINT AND DIFFERING IN DEGREE OF OVERWEIGHT (NARROW STRAW CONDITION)

By History	*By Current Weight*		
	Normal weight	*Overweight*	*Obese*
	Liked Milkshake (rating +1 to +3)		
At set point	2.33 (3)*	1.20 (5)	2.00 (2)
Below set point	1.50 (4)	1.50 (4)	1.33 (3)
	Disliked Milkshake (rating −3 to 0)		
At set point	−1.29 (7)	−2.60 (5)	−0.88 (8)
Below set point	−1.33 (6)	−2.00 (6)	−1.43 (7)

*Bracketed number represents the number of subjects in each category.

ings that extremely overweight subjects responded to taste like normals and unlike the moderately obese. Even in response to non-food external stimuli, the obese have been found to respond more like normals and less like their moderately overweight counterparts.[29] However our results do not support Nisbett's assertion that the moderately overweight group is most responsive to external cues because it is further from physiological set point. We question whether percent overweight is an adequate or even correct definition of deviation from set point. Weight stability appears to provide a conceptually more precise definition. When this definition is used, set point differences within weight groups had very little effect. They did not influence hedonic ratings at all and had no effect on intake when consumption involved but slight effort. The significantly decreased intake by below set point individuals using the effortful straw is subject to an explanation that is theoretically very trivial. Perhaps subjects who, by self report, try to consciously monitor their weight were reminded by the effort involved in using the thin straw that they should not be trying so hard to get this forbidden food. It does not appear that they were more upset or disturbed by the effort since their preference ratings were not significantly lower than those of the stable weight groups.

SUMMARY AND CONCLUSIONS

The moderately overweight do not show aberrant discrimination of sweet tastes nor do they show a greater preference for sweet than normals, at least with the test substances used. Rather than involving which specific stimuli are preferred, the differential taste responsiveness of the overweight appears as a heightened affective response to whatever taste substances they judge to be either hedonically positive or negative. Heightened responsiveness is expressed both in preference and ingestion. Consequently, responsiveness to food cues may simply be a special case of a much broader phenomenon of generalized stimulus sensitivity. In contexts unrelated to eating the obese have been found to be far more reactive than normal weight individuals to prominent external cues which affected their time perception [27] and ideation, emotionality and distractability.[26,30,32] Sweet tasting substances may thus be just one of many classes of stimuli to which overweight subjects are highly responsive. Certainly it is possible that this kind of overresponsiveness could play an important role in the development of obesity. Recently we demonstrated [31] that normal weight individuals with no history of overweight and who show a predisposition to respond to salient cues in their environment also show greater fluctuations in food intake and weight when placed in a novel and abundant food environment during an 8 week summer camp than other normal weight subjects.

In addition to suggesting that the difference between overweight and normals in taste lies in their affective responsiveness, our experiments also began to examine more closely the question of a biological set point in humans. It is difficult *a priori* to

specify each individual's set point and consequently to infer distance from it. The heuristic value of deviation from set point as a predictor for and explanation of eating and regulatory behavior is thus questionable although its descriptive value may be great. Moreover in human eating behavior, where habits and preference play so great a role, the long term regulation modified by set point may be overridden by environmental influences.[16] Several experiments have now demonstrated that the short and long term regulation of even normal weight people is greatly influenced by cognitive cues and habits at the expense of precise physiological regulation.[15,37,39,40,41] Increased responsiveness to external cues might, thus, contribute to overeating and weight gain. Whether extreme obesity somehow overrides or modifies hyperresponsiveness to external cues or is etiologically different remains an important question. Animal work points to multiple causation and numerous types of obesity; certainly this should also be expected for humans. This is speculation, however, and the critical experiments remain to be done. The answers lie in good, longterm prospective studies starting with newborns. The implications for an understanding of the development of obesity as well as for sweet preference, are considerable.

REFERENCES

1. Bray, G.A. and York, D.A.: Genetically transmitted obesity in rodents. *Physiol. Rev.,* 51:598–656, 1971.
2. Cabanac, M.: Physiological role of pleasure. *Science,* 173:1103–1107, 1971.
3. Cabanac, M. and Duclaux, R.: Obesity: Absence of satiety aversion to sucrose. *Science,* 168:469–497, 1970.
4. Cabanac, M., Minaire, Y., and Adair, E.: Influence of internal factors on the pleasantness of gustative sweet sensation. *Comm. in Behav. Bio.,* Part A, 1:77–82, 1968.
5. Corbit, J.D. and Steller, E.: Palatability, food intake, and obesity in normal and hyperphagic rats. *J. Comp. Physiol. Psychol.* 58:63–67, 1964.
6. Desor, J.A., Maller, O. and Turner, R.E.: Taste in acceptance of sugars by human infants. *J. Comp. Physiol. Psychol.* 84:496–501, 1973.
7. Fuller, J.L. and Jacoby, G.A.: Central and sensory control of food intake in genetically obese mice. *Am. J. Physiol.* 183:279–283, 1955.
8. Graff, H., and Stellar, E.: Hyperphagia, obesity, and finickiness. *J. Comp. Physiol. Psychol.* 55:418–424, 1962.
9. Grinker, J.: Obesity and Taste: Human and animal studies. In Bray, G.A. (Ed.), *Obesity in Perspective Part II,* Washington, D.C., Superintendent of Documents, U.S. Government Printing Office, 1976.
10. Grinker, J., Hirsch, J., and Smith, D.C.: Taste sensitivity and susceptibility to external influence in obese and normal weight subjects. *J. Pers. Soc. Psychol.* 22:320–325, 1972.
11. Hashim, S. and Van Itallie, T.: Studies in normal and obese subjects with a monitored food-dispensing device. *Ann. N. Y. Acad. Sci.* 131:654–661, 1965.
12. Herman, C.P. and Mack, D.: Restrained and unrestrained eating. Unpublished manuscript, Northwestern University, 1974.
13. Herman, C.P. and Polivy, J.: Effects of fear on restrained and unrestrained eaters. Unpublished manuscript, Northwestern University, 1974.
14. Hirsch, J. and Knittle, J.L.: Cellularity of obese and nonobese human adipose tissue. *Fed. Proc.* 29:1516–1521, 1970.

15. Jordan, H.A., Stellar, E., and Duggan, S.Z.: Voluntary introgastric feeding in man. *Comm. Behav. Bio.* 1:65–67, 1968.
16. LeMagnen, J.: Advances in studies on the physiological control and regulation of food intake. In Stellar, E. and Sprague, J.M., (Eds.), *Progress in physiological psychology, Vol. 4.* New York, Academic Press, 1971.
17. Metropolitan Life Insurance Company: New weight standards for men and women. *Stat. Bull.,* 40: 1–4, 1959.
18. Miller, N.E., Bailey, C.J., and Stevenson, J.A.F.: Decreased "hunger" but increased food intake resulting from hypothalamic lesions. *Science,* 112: 256–259, 1950.
19. Moskowitz, H.R.: The sweetness and pleasantness of sugar. *Am. J. Psych.* 84: 387–405, 1971.
20. Mrosovsky, N.: *Hibernation and the hypothalamus.* New York, Appleton-Century-Crofts, 1971.
21. Nisbett, R.E.: Taste, deprivation and weight determinants of eating behavior. *J. Pers. Soc. Psychol.* 10:107–116, 1968.
22. Nisbett, R.E.: Eating behavior and obesity in men and animals. *Adv. Psycho. Med.* 7:175–190, 1972.
23. Nisbett, R.E.: Hunger, obesity and the ventromedial hypothalamus. *Psych. Rev.,* 79:433–453, 1972.
24. Nisbett, R.E. and Gurwitz, S.B.: Weight, sex, and the eating behavior of human newborns. *J. Comp. Physiol. Psych.* 72:245–253, 1970.
25. Price, J.M. and Grinker, J.: Effects of degree of obesity, food deprivation and palatability on eating behavior of humans. *J. Comp. Physiol. Psych.* 85: 265–271, 1973.
26. Rodin, J.: Effects of distraction on the performance of obese and normal subjects. *J. Comp. Physiol. Psych.* 83: 68–75, 1973.
27. Rodin, J.: Causes and consequences of time perception differences in overweight and normal weight people. *J. Pers. Soc. Psych.* 31: 898–904, 1975.
28. Rodin, J.: The effects of obesity and set point on taste responsiveness and ingestion in humans. *J. Comp. Physiol. Psych.* 89: 1003–1009, 1975.
29. Rodin, J., Herman, C.P., and Schachter, S.: Obesity and various tests of external sensitivity. In S. Schachter and J. Rodin (Eds). *Obese humans and rats.* Washington, Erlbaum/Wiley, 1974.
30. Rodin, J. and Slochower, J.: Fat chance for a favor: Obese-normal differences in compliance and incidental learning. *J. Pers. Soc. Psych.* 29:557–565, 1974.
31. Rodin, J. and Slochower, J.: Externality in the nonobese: Effects of environmental responsiveness on weight. *J. Pers. Soc. Phych.* 33:338–344, 1976.
32. Rodin, J. and Singer, J.E.: Eye shift, thought and obesity. *J. Pers.,* in press.
33. Rytand, D.A.: Hereditary obesity of yellow mice: A method for the study of obesity. *Proc. Soc. Exper. Bio. Med.* 54:340–341, 1943.
34. Schachter, S. and Rodin, J.: *Obese humans and rats.* Washington, Erlbaum/Wiley, 1974.
35. Sclafani, A., Belluzzi, J.D., and Grossman, S.P.: Effects of lesions in the hypothalamus and amygdala on feeding behavior in the rat. *J. Comp. Physiol. Psych.* 72:394–403, 1970.
36. Sclafani, A., and Grossman, S.P.: Hyperphagia produced by knife cuts between the medial and the lateral hypothalamus in the rat. *Physiol. Behav.* 4:533–537, 1969.
37. Spiegel, T.: Caloric regulation of food intake in man. *J. Comp. Physiol. Psych.* 84:24–37, 1973.
38. Teitelbaum, P.: Sensory control of hypothalamic hyperphagia. *J. Comp. Physiol. Psych.* 48:156–163, 1955.
39. Walicke, B. C., Jordan, H.A., and Stellar, E.: Preloading and the regulation of food intake in man. *J. Comp. Physiol. Psych.* 68:327–333, 1969.
40. Wooley, S.C.: Physiologic versus cognitive factors in short-term food regulation in the obese and non-obese. *Psycho. Med.* 34:62–68, 1972.
41. Wooley, O.W., Wooley, S.C., and Dunham, K.B.: Can calories be perceived and do they affect hunger in obese and non-obese humans. *J. Comp. Physiol. Psych.* 80, 250–258, 1972.

42. Young, P.T.: Palatability: The hedonic response to foodstuffs. In Code, C.F. (Ed.) *Handbook of Physiology, Section 6, Alimentary Canal.* Washington, D.C. American Physiological Society, 1967, pp. 353–366.

DISCUSSION

PFAFFMANN: With regard to the milk shake study, what would you think of the interpretation that instead of the enhancement of the experience with the taste solution in the overweight, there is a reduction of the mouth satiety effectiveness in reducing appetite?

RODIN: That is quite possible. Cabanac's work [1,2,3] indicates that the high hedonic ratings which overweight subjects give to oral sucrose solutions do not drop after an oral glucose load while they show a dramatic decrease in normals. This suggests that, in the obese, taste for sweet is not affected by short-term satiety cues.

LE MAGNEN: Cabanac made some experiments showing that subjects after a weight loss by food deprivation, behave as obese subjects simply because they were, as obese, hungry. Relative palatabilities of food items are modified by hunger.

RODIN: Although Cabanac [1] shows that for the few normal subjects he tested, weight loss was related to a likelihood to respond more like obese subjects, neither we [4] nor Grinker (this volume) have found these results. Weight loss in many overweight and some normal weight subjects did not lead to increased responsiveness in sweet taste.

LE MAGNEN: It is the matter of degree. Obese, even at their maximum overweight, are still presumably below their set point and, therefore, in a situation comparable to a normal subject after a forced weight loss.

RODIN: I am not certain that we can assume that an obese person is below his set point. In general, degree of overweight seems a faulty metric for nearness to set point and as the data I have presented demonstrate using weight stability as a measure of set point, shows no taste responsiveness differences between subjects at or below set point. Hunger and deprivation certainly must influence responsiveness to sweet taste but one's degree of overweight may not be the crucial intervening factor.

[1] Cabanac, M.: Physiological role of pleasure. *Science* 173:1103–1107, 1971.

[2] Cabanac, M. and Duclaux, R.: Obesity: Absence of satiety aversion to sucrose. *Science* 168:496–497, 1970.

[3] Cabanac, M., Minaire, Y., and Adair, E.: Influence of internal factors on the pleasantness of gustative sweet sensation. *Communications in Behavioral Biology, Part A,* 1:77–82, 1968.

[4] Rodin, J.: The relationship between external responsiveness and the development and maintenance of obesity. In Novin, D., Wyrwicka, W. and Bray, G.A. (Eds.). *Hunger: Basic Mechanisms and Clinical Implications.* New York, Raven Press, 1976.

Chapter 22

EFFECTS OF METABOLIC STATE ON TASTE PARAMETERS AND INTAKE: COMPARISONS OF HUMAN AND ANIMAL OBESITY

Joel A. Grinker

During the last few years we have investigated the role of taste factors in human and animal obesity.[13,15] We asked four specific questions: Does the degree of obesity correlate with detection thresholds or preferences for sweet tastes? Do taste preferences change in response to short-term food deprivation? Does the degree of obesity influence taste preferences following short-term food deprivation? Finally, what is the most appropriate animal model for studying the eating behavior and taste response of obese humans? We compared taste responses in adult humans and children of normal weight with responses in adults and children of varying degrees of obesity. We examined three models of obesity in the rat: experimentally produced obesity following lesions of the ventromedial hypothalamus, experimentally produced obesity following over-nutrition in the preweaning period, and genetic obesity in the Zucker rat.

OBESITY AND "HEIGHTENED" TASTE RESPONSIVENESS IN RATS

Normal weight animals are susceptible to manipulations in the palatability of their diet. Thus, Sprague Dawley rats will drink sweetened water in preference to plain water [38] and may even become obese on exotic garbage or supermarket diets.[45] In the obese state "overeating" of highly palatable diets often occurs. Over-indulgence in "sweets" has even been singled out as a cause of human obesity.[36]

VMH Obesity

The major evidence for an association between taste responses and metabolic state has come from studies of animals made obese by lesions of the ventromedial hypothalamus (VMH). These animals show an increased intake of good tasting (high fat or high carbohydrate) diets and decreased intake of less palatable diets (e.g. adulterated with quinine). They may fail to exhibit hyperphagia and weight gain on less palatable diets and may lose weight if the palatability of the diet is further decreased.[8,25,44]

The role of taste or sensory factors

in hyperphagia, and the impaired food motivation of the VMH animal has been questioned. As early as 1962, Graff and Stellar [11] concluded that obesity was not a necessary condition for the finickiness associated with hypothalamic lesions. It is now known that both the hyperphagia and the impaired food motivation of the VMH animal can be reduced or eliminated by pretraining or reduction in body weight.[27,46,51] Several investigators have reported that the hyper-reactivity to taste stimuli can be reduced by pre-operative training or by separating the taste stimuli from the animal's diet.[2,28,32,47]

To explore the relation of taste to VMH obesity we made electrolytic lesions in the ventromedial hypothalamus of male Sprague Dawley rats and compared their responses to sucrose solutions to that of sham operated controls under a variety of feeding conditions.[18,41] Using a brief exposure technique that reduces the effects of post-ingestional factors (see ref. 6) we recorded responses to non-dietary sweet solutions in both one and two bottle paradigms. Table 22–1 describes the experimental procedures and animal characteristics. One set of controls and VMH animals fed lab chow *ad libitum* was considered the static group. Another set of controls, restricted to 75% of baseline intake and VMH lesioned animals restricted to pre-operative intake levels (approximately 75%) was considered the dynamic group. Hyperphagia and rapid weight gain occur immediately following VMH lesioning. The static phase is achieved when a plateau in body weight occurs and the hyperphagia is reduced or eliminated (see ref. 3). By restricting the animal's food intake, the dynamic phase can be extended indefinitely.

In the one bottle paradigm, animals deprived of liquids 1½ hours before testing were given access to solutions (.5, 1, 4, 8 and 32% wt./vol.) (.015, .029, .117, .234 and .934M) of sucrose for 30 minutes. This brief deprivation was sufficient to insure adaptation to the experimental procedures without producing fluid deprivation and consequent flattening of the preference function (see ref. 7). The drinking of VMH animals did not differ from that of their ad lib fed controls. Under 75% food restriction, however, the 30

TABLE 22–I

STUDIES OF
FOOD INTAKE AND OBESITY IN RATS

	Number	*Sex*	*Age*	*Weight*	*Food Intake (grams)*
Static phase of weight gain in Sprague Dawleys:					
VMH Lesioned	8	M	6 mo.	641.87	37.15
Sham-Operated	8	M	6 mo.	516.93	25.27
Dynamic phase of weight gain in Sprague Dawleys:					
VMH Lesioned	7	M	5 mo.	456.53	27.91
Sham-Operated	7	M	5 mo.	454.39	21.52
Genetic Obesity					
Zucker Fatties fa/fa	5	M	5 mo.	680.76	41.09
Zucker Leans Fa/	6	M	5 mo.	451.50	28.50

minute intakes of the VMH animals for all solutions decreased markedly (Anova: Static-Dynamic/VMH-Sham $F=21.2787$, $p<.005$) (See Figure 22–1)

In two bottle paradigms, sucrose solutions of differing concentrations were presented simultaneously for 30 minutes. For each pairing, all animals preferred the more concentrated solution and there were no differences in the preference responses of static VMH and their sham-operated controls. The dynamic VMH animals, however, showed a reduction in intake for the most concentrated solution and an increase in intake of the least concentrated. (Anova: Dynamic-Static/VMH-Sham $F=4.275$, $p<.005$) (See Figure 22–2)

In both one and two bottle paradigms static VMH animals and

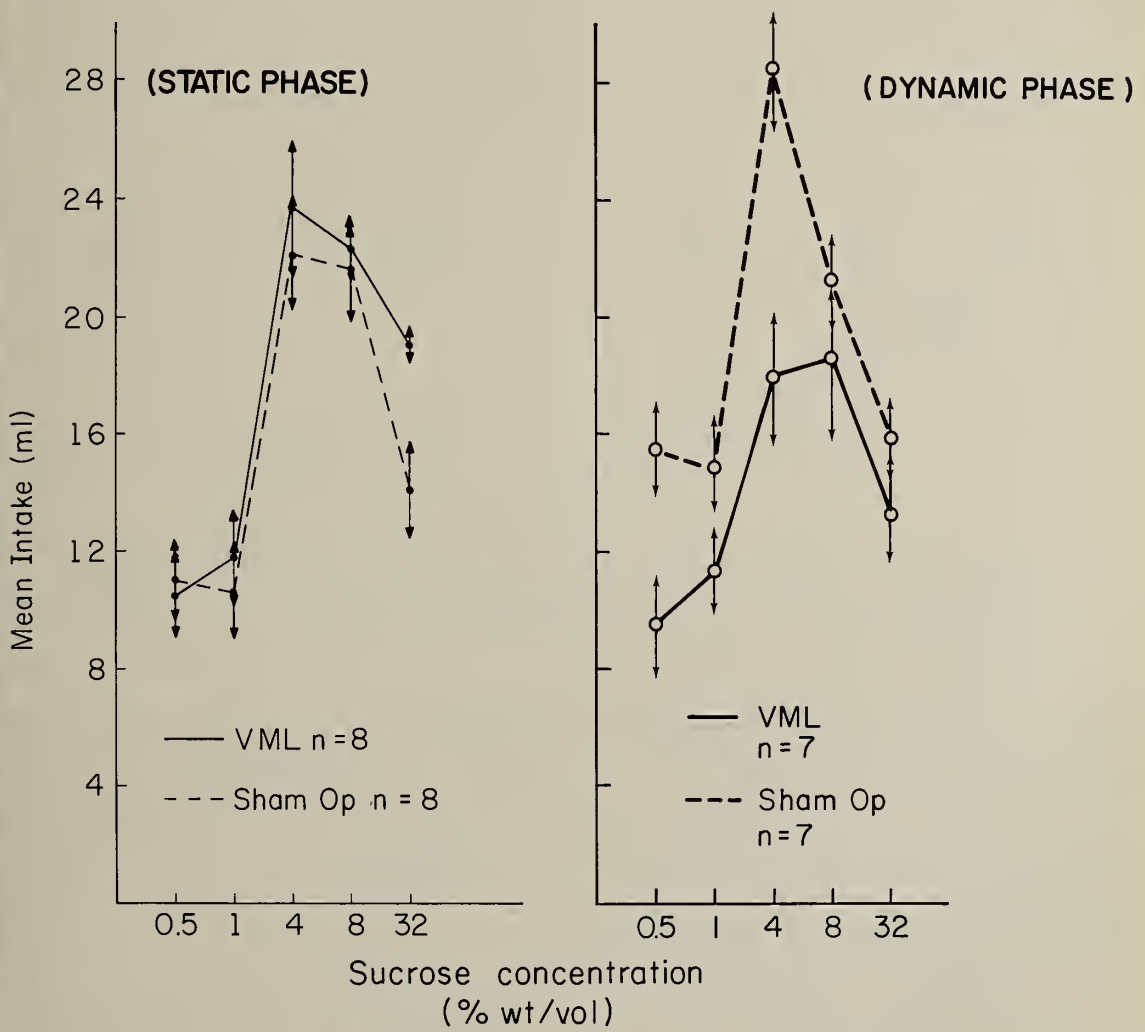


Figure 22–1. Mean ± (SEM) intake of sucrose solutions as a function of concentration by ventromedial hypothalamic lesioned (VML) and sham-operated Sprague Dawley rats under ad lib (static) or food-restricted (dynamic) maintenance conditions. Solutions were presented in a single bottle paradigm for 30 minutes.

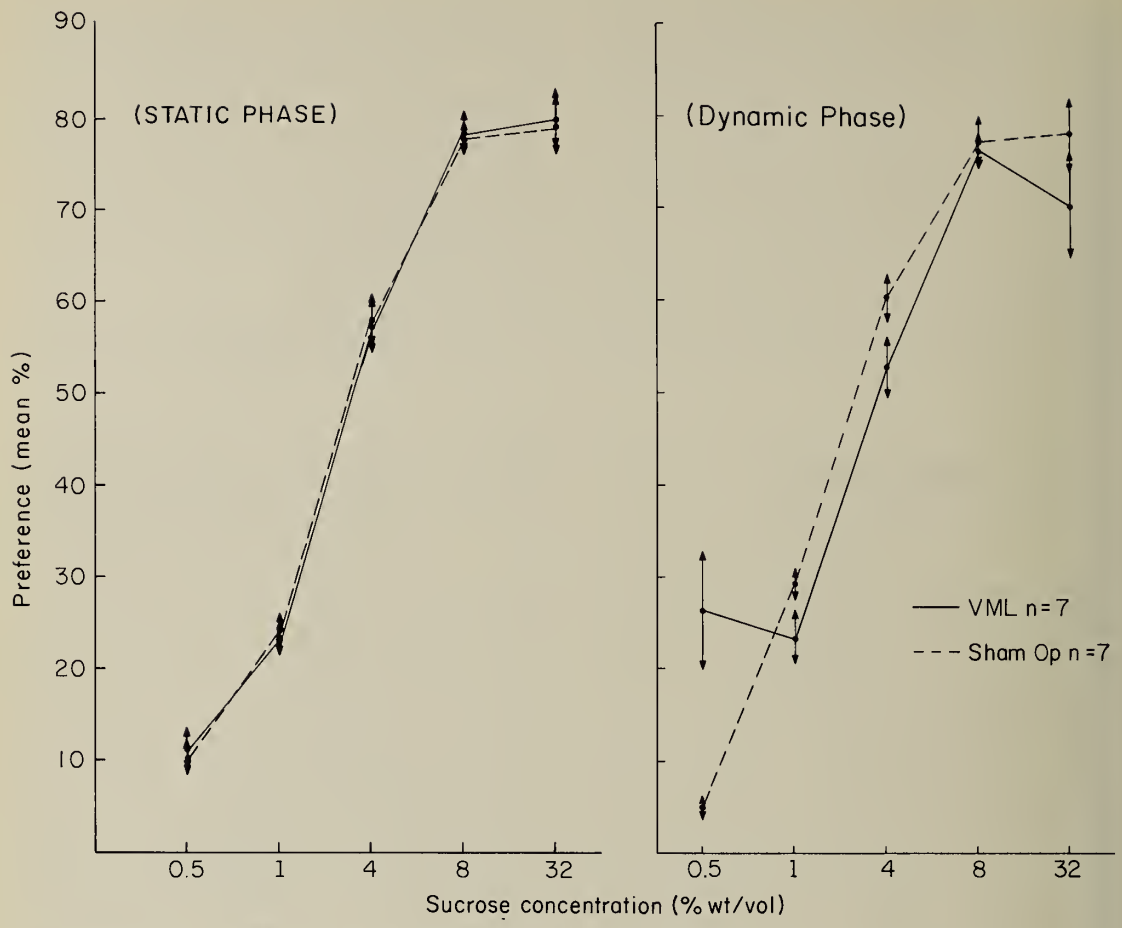


Figure 22–2. Mean percent preference for sucrose concentrations by ventromedial hypothalamic lesioned (VML) and sham-operated Sprague Dawley rats maintained on ad lib (static) or food-restricted (dynamic) diets. Pairs of sucrose solutions were presented for 30 minutes.

sham operated controls had equivalent intakes of sucrose solution. When food intake was restricted to preoperative levels, however, the sucrose drinking of VMH animals decreased. The results are contrary to earlier hypotheses that hyper-responsiveness to sweet solutions is present in both the dynamic and static VMH animals.[8] One major difference in the present experiments and others is that we measured the consumption of sweet solutions rather than the consumption of the animal's diet. The findings of hyper-responsiveness to good tastes would seem dependent not only on the presence of obesity and the lack of food deprivation, but also on the method of presentation of test stimuli.

Genetic obesity

Unlike animals that become obese after VMH lesions, genetically obese Zucker rats appropriately adjust food intake for calories when confronted with high fixed ratio schedules, caloric

dilution or quinine adulteration.[9, 12] Differences between these two animal models of obesity are not only behavioral but also metabolic (see ref. 15, 49). The increased adiposity of the VMH animal is accomplished solely through an increase in adipose cell size without an increase in cell number.[21] In contrast, genetically obese Zucker rats have increases in both adipose cell number and size.[21]

We studied the taste responses of the genetically obese Zucker rat (fa/fa) using experimental procedures similar to those reported above.[41] Table 22–I outlines relevant animal characteristics and procedures. In the one bottle paradigm obese Zucker rats showed no difference in sucrose solution drinking compared to their lean littermates (see Figure 22–3). When given a choice between two sweet solutions, the obese Zucker rats showed a less pronounced preference than their lean littermates (Anova: fat-lean/sol $F = 5.1497$, $p < .01$) (see Figure 22–4) Again, obesity did not potentiate or heighten taste responsiveness. These experiments suggest that obesity is uncorrelated and independent of taste responsiveness.

Experimental obesity produced by over-nutrition

The taste responses of heavy and light male and female Sprague Dawleys from small and large litters were compared at seven weeks of age (Johnson, Greenwood and Cruce, unpublished data). All animals showed a preference for 0.25% saccharine over 3% glucose, with heavy female animals showing a greater preference for saccharine than light females in both the *ad lib* and the food restricted conditions. These differences in taste responses may reflect differences in metabolic state. Knittle and Hirsch [26] have reported that over-nutrition during the pre-weaning period (animals raised from birth to weaning in small litters) produces significant increases in weight and adipose cell size and number compared to under-

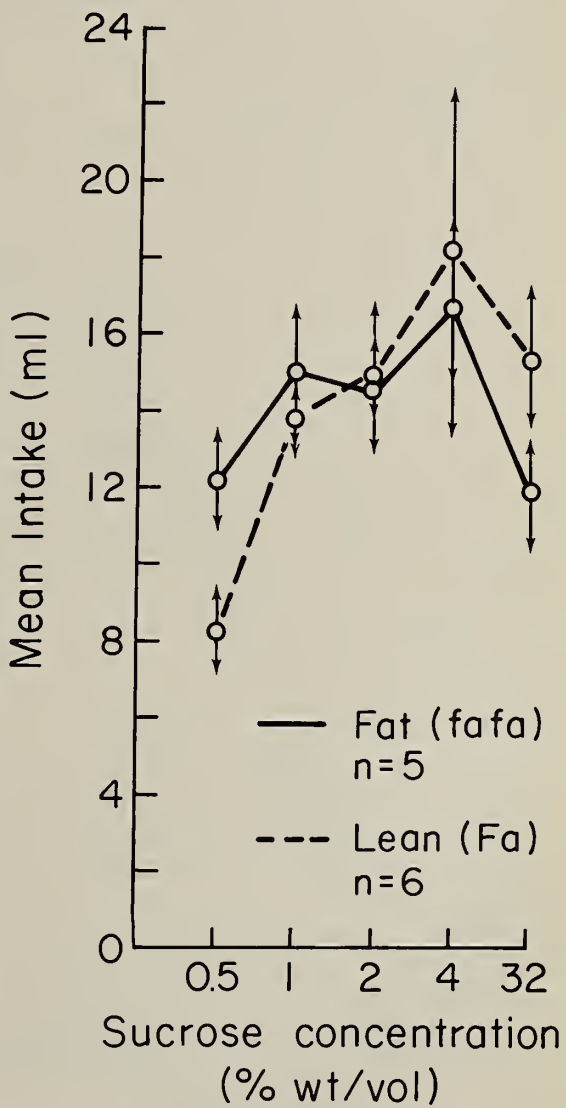


Figure 22–3. Mean ± (SEM) intake of sucrose solutions as a function of concentration by Zucker obese (fa/fa) and non-obese (Fa/–) rats during a 30 minute access to a single concentration.

nutrition during the pre-weaning period (animals raised in large litters) and that these differences persist into adult life. The present experiment suggests that early rearing experience can produce persistent changes in behavior as well as in body size and adipose cellularity. The results are dramatic for the females but the males fail to show a differential taste responsiveness as a function of weight. Thus, another factor (sex) would seem to modulate any link between obesity or degree of overweight and taste responsiveness.

ARE OBESE HUMANS LIKE OBESE RATS?

Responsivity to feeding cues

In studies of human obesity it is difficult to distinguish causation from correlation since the behavioral and metabolic pathology found in the obese reflect the combined effects of genetic predisposition, cultural influences and other environmental factors. However, relative overeating by moderately obese subjects when food is freely available and attractive coupled with relative undereating by these subjects when food is unattractive or effort is required [19, 33, 34, 42] has led Schachter [43] to a provocative hypothesis. He suggests that the elevated food intake in obese relative to normal weight humans results from their greater responsiveness to external cues (such as sight, smell and time of day) and lesser responsiveness to "internal" physiological cues. But, in our studies, manipulating the degree of hunger failed to produce the corresponding variation in amount of ingestion that this hypothesis implies should characterize the response of normal weight subjects. Instead both obese and normal weight subjects were unresponsive to satiety signals and consumed equivalent amounts under conditions of deprivation or non-deprivation. Specifically, there was no difference between the number of crackers eaten before and after lunch for either obese or normal weight subjects participating in what

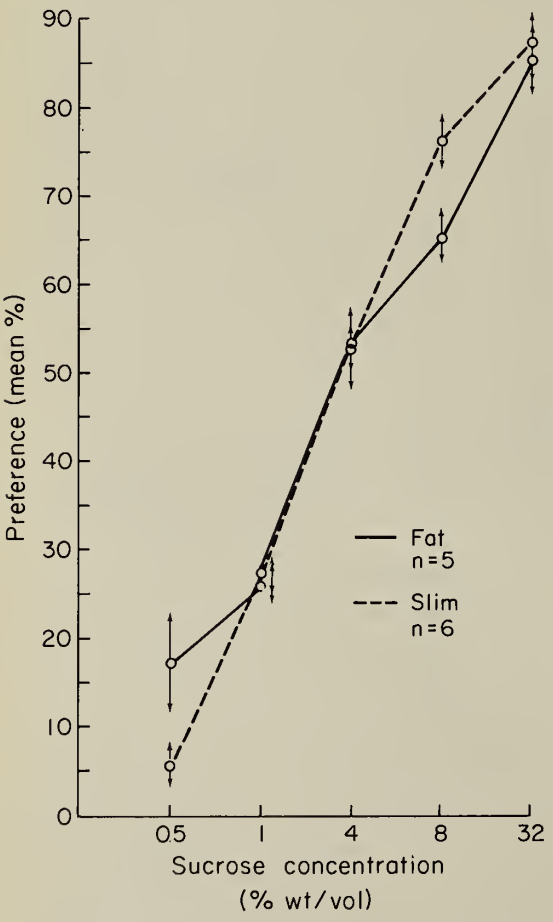


Figure 22–4. Mean percent preference for sucrose concentrations by Zucker obese and non-obese rats. Pairs of sucrose solutions were presented for 30 minutes.

they were told was a study of sensory and taste qualities of different varieties of crackers.[40] The covertly recorded data are presented in Figure 22–5. The subject characteristics are given in Table 22–II, Experiment A.

A more serious challenge to hypotheses linking obesity to responsiveness to external cues is the finding that the eating behavior of all subjects (including normal weight subjects) can be controlled by external or cognitive cues.[14, 22, 48, 52, 53]

Since the testing situation may have quite different meanings for obese and normal weight subjects it is unclear to what degree any excessive reliance on external or sensory cues by the obese, even in a covert experiment, can be interpreted as a causal factor in obesity. The reported externality of the obese may be a deliberate shift to reliance on external cues rather than an inability to sense internal cues or a denial of their importance.

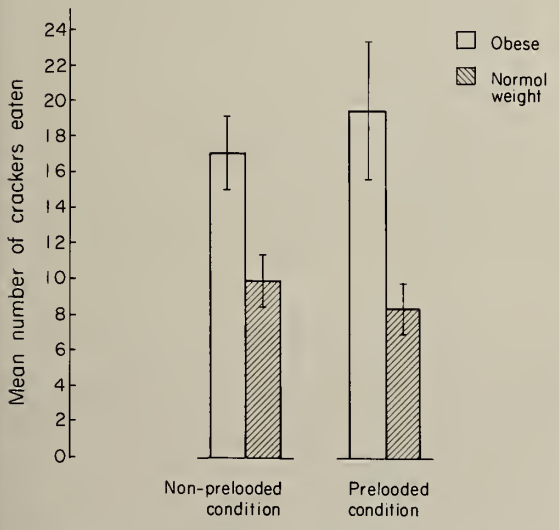


Figure 22–5. Mean number ± (SEM) of crackers eaten during taste testing by obese and normal weight subjects as a function of a sandwich pre-load (before and after lunch).

Stunkard and Koch[50] correlated gastric motility and self reported hunger in obese and normal weight subjects. Gastric motility was continuously recorded over a four hour period following an overnight fast. Every 15 minutes subjects reported their hunger. Using a signal detection analysis, it was possible to separate sensitivity or the ability to detect motility from criterion or willingness to report hunger. Both obese and normal weight subjects reported feeling hungry only 38% of the time when no contractions were present. However, normal weight subjects double their reports of hunger during the presence of contractions whereas obese subjects failed to increase their reports. This denial of internal cues (here gastric contractions) by the obese can be clearly separated from their perception of these cues.

The above experiments may provide an alternative explanation for the reported hyperresponsivity of obese individuals to external cues. In the presence of severe hunger, obese subjects may well be utilizing a strategy to keep themselves in control and avoid the temptation to overeat. Under conditions of high food deprivation obese subjects may deny or ignore food stimuli. This alternative explanation explains the puzzling observation that normal weight subjects increase their grocery purchases as a function of hours of food deprivation while obese subjects actually decrease their purchases with increasing hours of deprivation.[35]

Intake measurement and taste response

In our efforts to circumvent the so-

TABLE 22-11
SUBJECT CHARACTERISTICS

Experiment	Group	M	F	Age	Wt. (Kg.)	% Overweight
	ADULTS					
A. Test of Externality-Internality Hypothesis: Intake of Crackers Before and After a Meal	Extremely Obese Long Term In-Hospital Weight Reduction Patients	6	14	32.8	140.9	122.3
	Normal Weight Volunteers	6	14	25.4	60.6	-3.5
B. Sucrose Detection and Magnitude Estimation	Extremely Obese Enrolled in an In-Hospital Weight Reduction Program	14	25	35.8	121.3	105.0
	Normal Weight Volunteers	9	4	23.0	70.1	8.0
C. Sucrose Taste Preference: Hedonic Ratings, Paired Comparison and Consumption of Sucrose	Extremely Obese Outpatients	9	16	34.2	136.0	125.2
	Moderately Obese Outpatients	3	11	32.7	93.4	45.8
	Normal Weight Volunteers	7	10	23.1	63.5	0.0
D. Sucrose Taste Preferences Before and After Weight Reduction	Extremely Obese Inpatients 10 Month Hospitalization	8	1	25.4	162.2[a] 101.0	125.0
	Extremely Obese Inpatients 3 Month Hospitalization	2	3	30.6	132.2[a] 115.7	200.0
E. Sucrose Taste Preferences Before and After a Glucose Load	Normal Weight Volunteers	7	7	24.4	67.6	0.0
	ADOLESCENTS AND CHILDREN					
F. Sucrose Taste Preference Paired Comparisons	Children at Outpatient Weight Reduction Clinic	3	5	8-10	51.8	50.0
	Moderately Overweight Children in School	6	2	8-10	40.5	25.0
	Normal Weight Children in School	5	5	8-10	25.7	1.0
G. Sweetened Kool-Aid Taste Preference: Before and After Weight Reduction, Before and After a Meal	Obese Adolescents at Summer Camp for Weight Reduction	-	14	14-16	79.9 69.7	43.2

Note a: Testing was carried out at the original and reduced weights while subjects were on isocaloric diets.

cial and cognitive expectations that the obese bring to an eating situation, we have examined taste responses in several animal models of obesity. Next, we examined taste parameters independent of food intake in obese and normal weight adults and children using appropriate psychophysical procedures.[13,15,16,17] Using a criterion-free signal detection procedure we found no differences in the ability to detect low concentrations of sucrose between obese and normal weight subjects. The weight, age and sex characteristics of the test population is given in Table 22–II, Exp. B. We next manipulated the perception of sweetness by the addition of a red "cherry" color. With a magnitude estimation procedure, all subjects rated suprathreshold solutions of sucrose colored red more sweet than colorless solutions of the same concentration. Thus they demonstrate equivalent responsivity to an external cue. We did, however, obtain marked differences in taste preference. Normal weight subjects preferred the concentration of medium strength (a pattern similar to that reported by Pfaffmann [39]) whereas the preferences of obese subjects were an inverse function of concentration; the more concentrated the solution, the less it was preferred (see Table 22–II, Exp. C).

Sweet aversion in obese humans

The differences between obese and normal weight subjects in taste preferences were found to be specific to the sweet taste.[13, 15] Both obese and normal weight subjects showed pronounced aversions for NaCl which were similar to the aversion pattern reported by Pfaffmann.[39] The findings of a sucrose aversion would appear to be independent of experimental procedures or psychophysical methodology, since we have obtained similar results with hedonic ratings and paired comparison procedures (See Figures 22–6 and 7). Degree of obesity was correlated with sucrose aversion; moderately overweight subjects disliked the sweet solutions less than extremely obese subjects. The sucrose aversion function of the obese was responsive to manipulations which affect palatability such as temperature; a lower temperature decreasing the rated aversiveness, a higher temperature increasing the rated aversiveness.

The question of validity is more

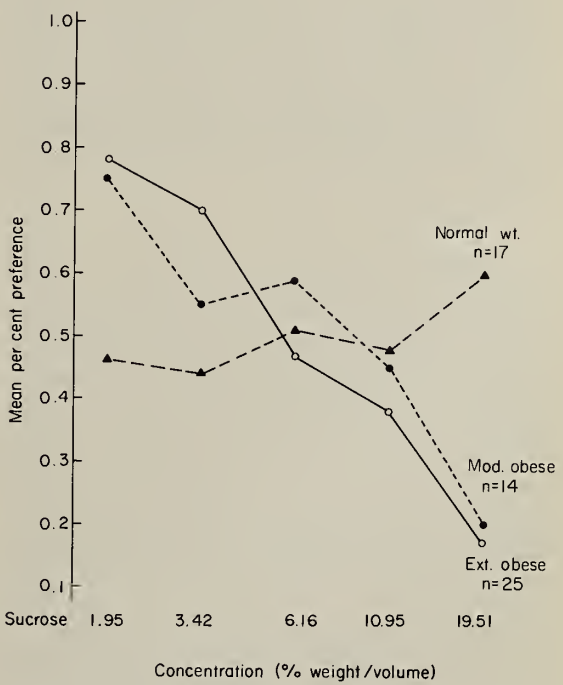


Figure 22–6. Sucrose preferences of extremely obese, moderately obese and normal weight subjects using the Paired Comparisons procedure. Percent preference reflects the number of times a solution was chosen over all other solutions.

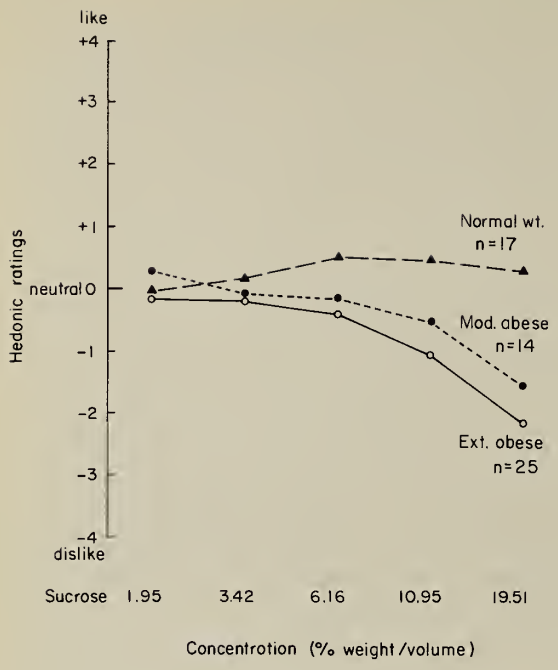


Figure 22–7. Hedonic ratings of sucrose solutions as a function of concentration by extremely obese, moderately obese and normal weight subjects. Single concentrations of sucrose were presented in random order. Subjects tasted each solution without swallowing, rinsed with distilled water and indicated their liking.

complex. Animal preferences are based on measures of actual intake. In humans, measures of preference are based on the assumption that hedonic ratings or other verbal responses accurately reflect the intake of foods. One solution to the problem is to measure directly the ingestion of different sucrose solutions by obese and normal weight subjects and to correlate intake with preference ratings. We told subjects that we were interested in the thirst quenching properties of various solutions and asked them to refrain from drinking any liquid for two hours before testing. Subjects then received 1 of 5 possible sucrose concentrations to drink and rate at each test session. Consumption was highly correlated with taste preferences not only for normal weight, but also for moderately overweight and severely obese subjects (see Figures 22–8 and 9).

How well do such measures obtained with sucrose solutions correlate with preference for actual foods? Aside from anecdotal statements, we have no data on our obese subjects' actual food choices; subjects reported overeating foods such as meat, salads and bread but little interest in sweets. Other experimenters have confronted the same issue. Moskowitz [31] using magnitude estimation techniques and category scaling examined the intensity and pleasantness of various sucrose concentrations as well as

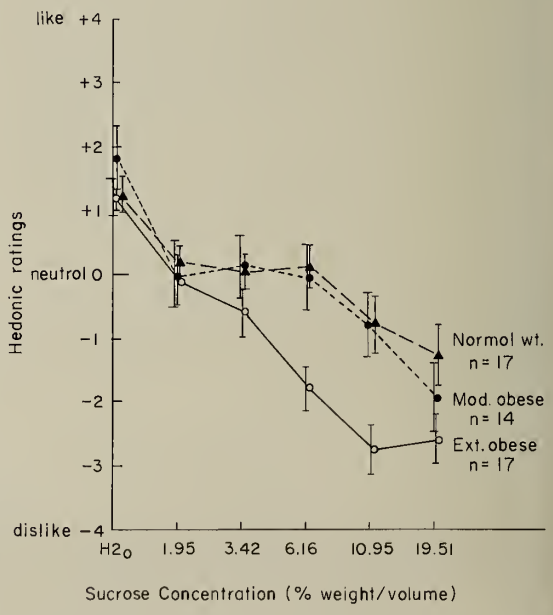


Figure 22–8. Hedonic ratings of sucrose solutions as a function of concentration by extremely obese, moderately obese and normal weight subjects after consumption. Subjects consumed as much as they desired of a single sucrose concentration for 10 minutes and then indicated their liking.

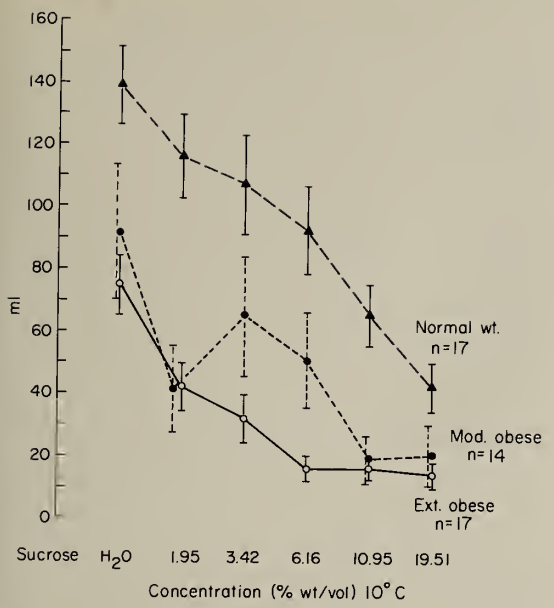


Figure 22–9. Amount consumed ± (SEM) of sucrose solutions as a function of concentration by extremely obese, moderately obese and normal weight subjects. Subjects consumed as much or as little as they desired of a single sucrose concentration. Order of concentration was randomized over days.

foods varying in sweetness in normal weight subjects. Sucrose preferences were significantly correlated with preferences for samples of solid foods of varying sweetness. These results strongly suggest that sucrose preferences measured by magnitude estimation or category scaling do generalize to sample foods and by inference, to actual food intake.

The finding of an aversion to sucrose solutions in the obese is contrary to the popular belief that the obese have a "sweet tooth." We find this sucrose aversion consistently. Since this is the only taste parameter which is predictably different in the obese and since it is correlated with the degree of obesity one might ask whether the ontogeny of the sweet aversion and the ontogeny of the obesity are related. Accordingly, we examined the sweet preferences of obese and normal weight children.

Taste preferences in children

Taste preferences for varying concentrations of sucrose (1.95 to 19.5%) (.057—.570M) in normal weight and obese children ages 8–10 were measured by the method of paired comparisons.[7] Normal weight and moderately overweight children were students in a Civics Class in San Diego. Obese children were selected from those referred to the Mount Sinai Pediatric Clinic for treatment of obesity and were seen on their initial visit to the Clinic. Table 22–II, exp. F outlines subjects' characteristics. The taste responses of the obese children were strikingly similar to those of obese adults: the more concentrated the solution the less frequently it was preferred. The normal and moderately overweight children responded more like normal weight adults preferring the most concentrated solutions. No sex differences were found (Anova: Wt groups/solutions $F=6.2493$, $p<.005$). (see Figure 22–10)

TASTE PREFERENCE AND METABOLIC STATE

Adults

Early psychophysical studies had reported that hunger produced a lowered threshold for sucrose.[10,54] Recently several investigators [30] using signal detection procedures separated detectability or sucrose threshold from criterion or response bias. They

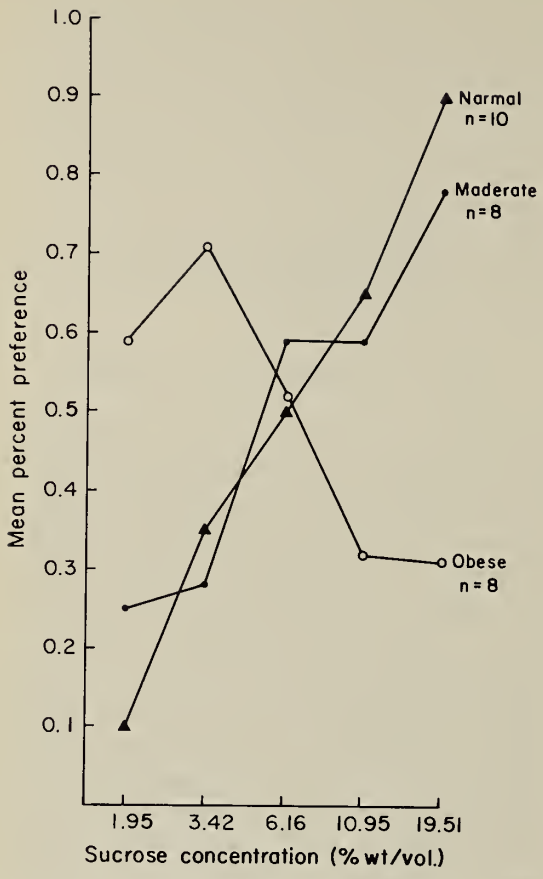


Figure 22–10. Sucrose preferences by extremely obese, moderately obese and normal weight children ages 8–10 using the Paired Comparisons Procedure. Percent preference reflects the number of times a solution was chosen over all other solutions.

concluded that subjects were more willing to report tasting sucrose prior to a meal than after. Other investigators have reported differences in preference as a function of metabolic state.[1,29]

The most recent attempts to correlate taste preference and metabolic state stem from the work of Cabanac.[4] Normal weight human subjects experience sweet solutions as unpleasant when tasted after a glucose load.[5] These results are interpreted as a consequence of changes in internal state. Along with H. Jordan, I attempted to replicate these phenomena in normal weight subjects,[13] but was hampered by substantial inter- and intra-subject variability (see the discussion by Stellar in this volume). Although the phenomenon of reduced pleasantness is present in normal weight subjects following a glucose load, only half the subjects actually showed the reduction in taste pleasantness (see Figure 22–11). When subjects were retested (either three days or three months later) no consistent response patterns emerged. Approximately half the subjects of each type (those initially showing the reduced pleasantness or not showing the reduced pleasantness) switched their response. The distribution of consistent and inconsistent subjects is given in Table 22–III, the subject characteristics in Table 22–II, exp. E.

EFFECTS OF LONG-TERM DEPRIVATION (WEIGHT REDUCTION) ON TASTE PARAMETERS

Data on the effects of weight reduction on taste preferences for sucrose concentrations (1.95%—19.5%) (.057—.570M) were obtained from adult subjects who were either undergoing long-term hospitalization for weight reduction or losing weight as outpatients (Table 22–II, exp. D). The nine subjects in one hospitalized group were on liquid diets high in carbohydrate or fat during the maintenance periods preceding and following weight reduction. Not only did the two diets fail to produce significant

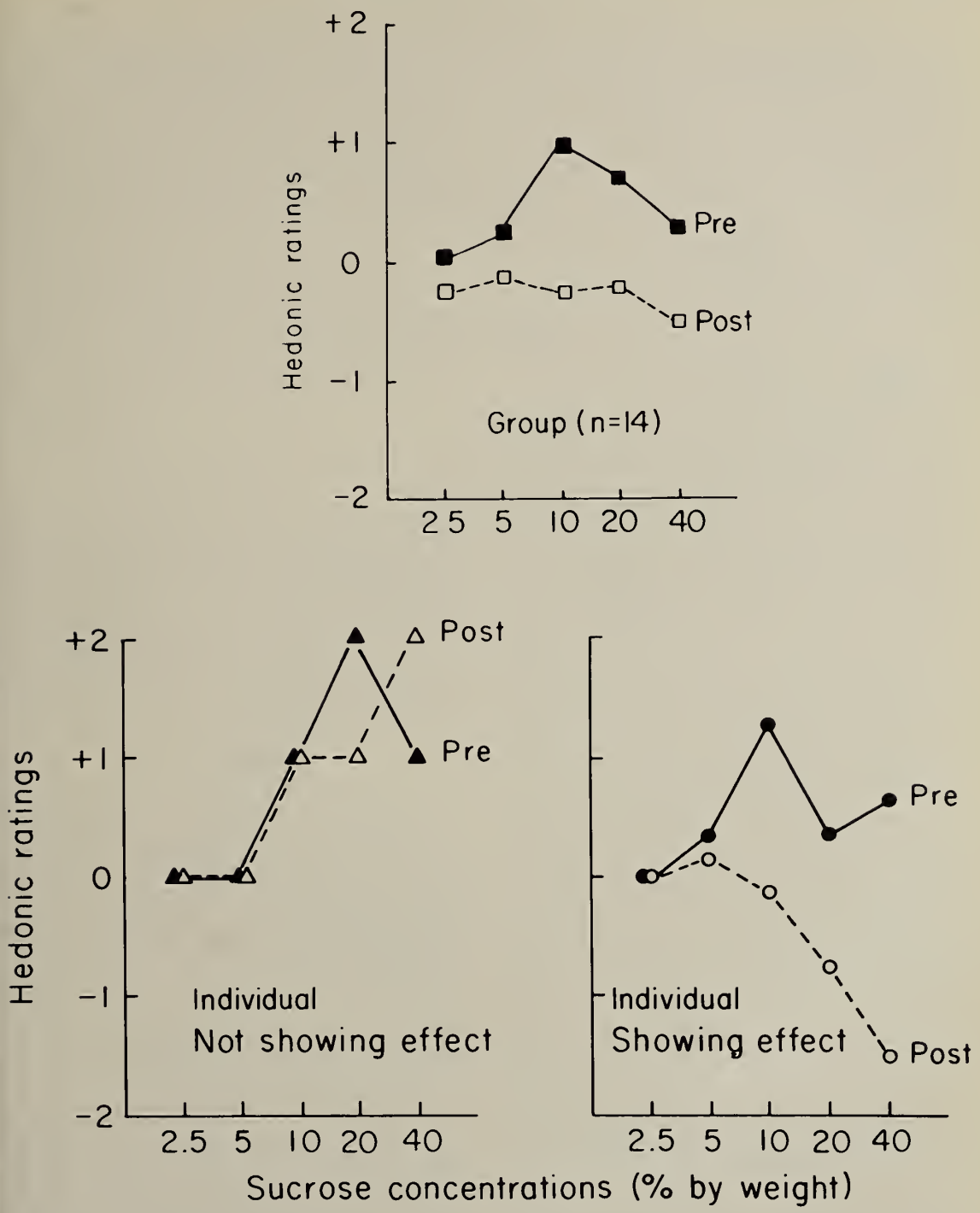


Figure 22–11. Mean hedonic ratings of sucrose solutions as a function of concentration by normal weight subjects before and after oral glucose loads. Group data (n = 14) are shown above. Individual data from two subjects are shown below; one subject shows a reduction in pleasantness following a glucose load, while one subject does not.

TABLE 22–III

RELIABILITY OF THE EFFECT OF GLUCOSE INGESTION UPON SWEET PREFERENCE JUDGEMENTS AT 72 HOURS RETEST OR 3 MONTH RETEST

Status of effect		*Test to retest interval*		*Combined sample*
at test	*at retest*	*72 hour*	*3 month*	
		Consistent subjects		
present	present	3	0	3
absent	absent	4	5	9
		7	5	12
		Inconsistent subjects		
present	absent	4	3	7
absent	present	2	1	3
		6	4	10

Note: The effect is initially present in almost half of the samples, present in 10 and absent in 12 subjects.

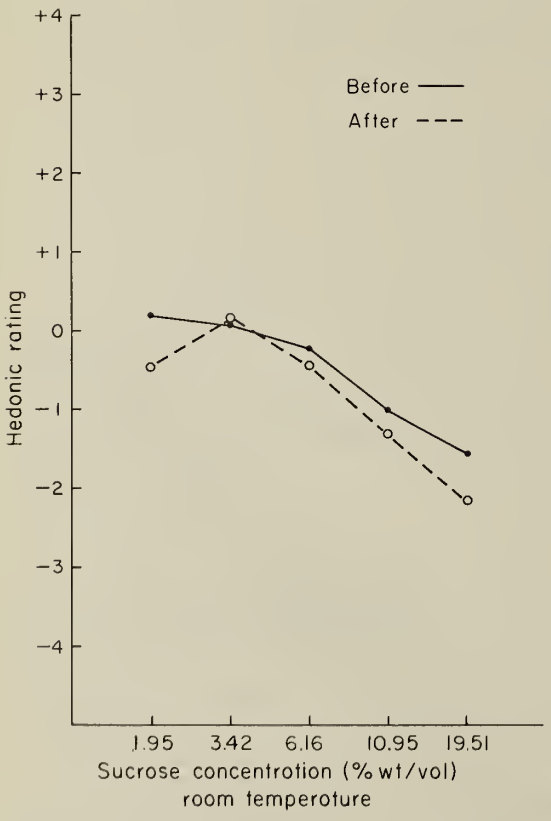


Figure 22–12. Mean hedonic ratings of sucrose solutions as a function of concentration by 14 extremely obese subjects before and after weight reduction. Subjects were tested on isocaloric diets before and after long-term in hospital weight reduction.

differences in taste preferences but weight reduction itself was without significant effect (see Figure 22–12). Outpatients and patients hospitalized for brief periods similarly failed to change their taste responses following weight reduction.

Children

Short-term changes in childrens' metabolic state consistently fail to affect their taste responses. Normal weight children did not show any differences in the percent preference for sucrose solutions (1.9 to 19.5%) when tested before and after lunch. Obese adolescents tested before and after dinner also did not change the rated attractiveness or percent preference for sweetened Koolaid solutions (0.5, 1.5, 2 x recommended sweetness).[17] Long-term changes like weight reduction, however, produced a significant effect in obese adolescents as the rated attractiveness of Koolaid solutions rose following weight reduction.[17] See Figure 22–13 and Table 22–II, Experiment G.

SUMMARY AND CONCLUSION

In humans, the degree of obesity is unrelated to sucrose detection thresholds, but is correlated with an aversion to sweet tastes. In rats, single or two bottle presentations of sweet solutions distinct from the diet do not elicit hyperresponsiveness or increased intake in the VMH animal. In contrast, the genetically obese rat does display a reduced preference for sweet solutions compared to its lean littermate. The cellularity pattern, normal glycemic response, and behavioral response to effort or diet adulterants of the genetically obese Zucker rat, make it an appropriate model for human obesity. The similarity between the taste responses of this animal and the obese human provides additional support for the usefulness of this model.

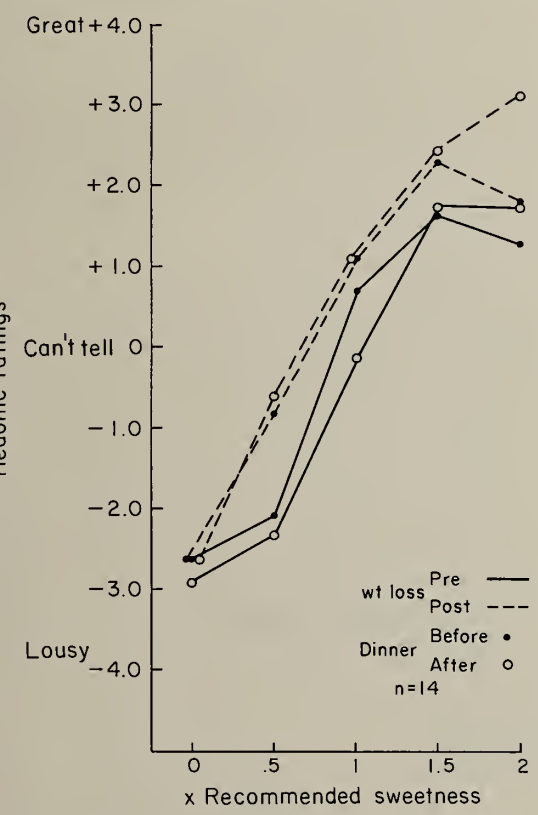


Figure 22–13. Mean hedonic ratings of Koolaid solutions (0, .5, 1, 1.5, 2 x recommended sweetness) by obese adolescents before and after a meal and prior to and after weight reduction. Sweetness was varied through the addition of 0, 12, 24, 36, or 48 ml of Sucaryl per gallon of spring water respectively. Subjects were attending a summer camp for weight reduction.

The popular myth that obesity is created by an over indulgence in "sweets" or carbohydrates persists although there are few direct observations or questionnaire studies comparing the food preferences and intakes of obese and normal weight subjects. In one study, no differences were found between obese and normal weight subjects at a county fair in their preferences for desserts of varying sucrose concentrations.[37] The self reports by obese teenagers showed a lower consumption of bread, sweets and sugar than those of normal weight children.[23] Similar findings of reported lower carbohydrate intake have been obtained from obese adults.[24] Since self reports are open to errors of denial or underestimation, other techniques for assessing food intake are needed. The lack of significant differences in fatty acids of adipose tissue between obese and normal weight subjects provides evidence that obese individuals eat the same proportion of fats, carbohydrates and protein as normal weight subjects do.[20]

Our findings of decreased liking for sweet solutions by both obese children and obese rats, have strengthened the view that the sucrose taste aversion seen in obese adults is a meaningful

phenomenon. Preliminary data suggest that this sucrose aversion can be elicited as early as three years of age. What are the implications for our understanding of obesity that fat children, fat adults and fat rats do not especially like sweets?

Although diet palatability may be a factor in overeating, we would conclude that it is not a major factor in the etiology or maintenance of the obese state. Since children who are obese at an early age show hypercellularity as well as hypertrophy of their adipose depots, it is interesting that the taste differences occur so early. It may be that early dietary intake has an important role in both the ultimate adipose tissue cellularity and the aversion to sucrose of obese children, adolescents and adults. At present, we have no data that suggest that adults who become obese late in life show differences in taste preferences from those who become obese early in life. Additionally, we do not know the effects of early nutritional treatments on taste preferences in humans. We have preliminary evidence that suggests that a highly palatable diet (Borden's sweetened condensed milk) reduces the intake of sucrose solutions in the genetically obese Zucker rat and not in its lean littermate. We also have data that suggest that early overnutrition (decreased litter size) significantly alters taste preference in females, but not in male rats.

The question of whether short or long-term food deprivation or restriction produces reliable differences in taste preferences remains unanswered. Our experimental results suggest that only by employing trained and experienced subjects can the significant inter- and intra-subject variability be reduced. Human psychophysical experiments often use trained observers. It may prove difficult, however, to integrate the need for experienced subjects with any covert experimental manipulations. Perhaps, rigid monitoring of the subjects' food intake prior to testing may reduce variability.

Weight reduction may reduce or eliminate the obese human's sucrose taste aversion. If these preliminary findings are substantiated, the study of taste preferences will have a direct usefulness for the study of obesity. We are beginning to study taste preferences in infants and children at high risk for obesity. I think that the essential questions are not what overweight individuals eat, but why they eat more, and how measures of taste preference relate to food intake.

REFERENCES

1. Bartolovic, B.: Internal regulation of gustatory sensitivity. *Acta Instituti Psychologici Universitatis Zagrabiensis*, No. 35–48, 73–80, 1964.
2. Beatty, W.W.: Influence of type of reinforcement on operant responding by rats with ventromedial lesions. *Physiol. Behav.*, 10:841–846, 1972.
3. Becker, E.E. and Kissileff, H.R.: Inhibitory controls of feeding by the ventromedial hypothalamus. *Amer. J. Physiol.*, 226:383–395, 1974.
4. Cabanac, M.: Physiological role of pleasure. *Science*, 173:1103–1107, 1971.
5. Cabanac, M., Minaire, Y. and Adair, E.R.: Influence of internal factors on the pleasantness of a gustative sweet sensation. *Comm. Behav. Biol.*, Part A, 1, 77–82, 1968.
6. Cagen, R.H. and Maller. O.: Brief exposure single-stimulus behavioral method. *J. Comp. Physiol. Psychol.*, 87:47–55, 1974.

7. Collier, G. and Bolles, R.: Some determinants of intake of sucrose solutions. *J. Comp. Physiol. Psychol.*, 65:379–383, 1968.
8. Corbit, J.D. and Stellar, E.: Palatability, food intake, and obesity in normal and hyperphagic rats. *J. Comp. Physiol. Psychol.*, 58:63–67, 1964.
9. Cruce, J.A.F., Greenwood, M.R.C., Johnson, P.R. and Quartermain, D.: Genetic versus hypothalamic obesity: Studies of intake and dietary manipulation in rats. *J. Comp. Physiol. Psychol.*, 87: 295–301, 1974.
10. Goetzl, F., Ahokar, A. and Payne, J.: Occurrence in normal individuals of diurnal variation in acuity in the sense of taste for sucrose. *J. Appl. Physiol.*, 2:619–626, 1950.
11. Graff, H. and Stellar, E.: Hyperphagia obesity and finickiness. *J. Comp. Physiol. Psychol.*, 55:418–424, 1962.
12. Greenwood, M.R.C., Quartermain, D., Johnson, P.R., Cruce, J.A.F. and Hirsch, J.: Food motivated behavior in genetically obese and hypothalamic hyperphagic rats and mice. *Physiol. Behavior*, 13:687–692, 1974.
13. Grinker, J.: Taste parameters in obese and normal weight individuals. Presented at Nutrition and Psyche. Eighth International Meeting, Sponsored by Fondazione Sasso, (in press).
14. Grinker, J.: Obesity and Taste: Human and animal studies. In Bray, G.A. (Ed.), *Obesity in Perspective Part II*, Washington, D.C., Superintendent of Documents, U.S. Government Printing Office, 1976.
15. Grinker, J. and Hirsch, J.: Metabolic and behavioral correlates of obesity. In Knight, J. (Ed.), Physiology, Emotion and Psychosomatic Illness. A CIBA Foundation Symposium 8 (new series), ASP, Amsterdam, 1972.
16. Grinker, J., Hirsch, J. and Smith, D.V.: Taste sensitivity and susceptibility to external influence in obese and normal weight subjects. *J. Pers. Soc. Psychol.*, 22:320–325, 1972.
17. Grinker, J., Price, J.M. and Greenwood, M.R.C.: Studies of taste in childhood obesity. In Novin, D., Wyrwicka, W. and Bray, G.A.: (Eds.) *Hunger: Basic Mechanisms and Clinical Implications.* New York, Raven Press, 1976.
18. Grinker, J., Roswell, V. and Ball, G.: A comparison of taste responsiveness in two animal models: Ventromedial hypothalamic lesioned and genetically obese rats. (Submitted for publication).
19. Hashim, S.A. and Van Itallie, T.B.: Studies in normal and obese subjects with a monitored food dispensing device. *Ann. N.Y. Acad. Sci.*, 131:654–661, 1965.
20. Hirsch, J. and Goldrick, B.: Serial studies on the metabolism of human adipose tissue. I: Lipogenesis, free fatty acid uptake and release in small aspirated samples of subcutaneous fat. *J. Clin. Invest.*, 43:1176–1792, 1964.
21. Johnson, P.R., Zucker, L.M. Cruce, J.A.F. and Hirsch, J.: Cellularity of adipose depots in the genetically obese Zucker rat. *J. Lipid Res.*, 12:706–714, 1971.
22. Jordan, H.A., Stellar, E. and Duggan, S.Z.: Voluntary introgastric feeding in man. *Comm. Behav. Biol.*, 1:65–67, 1968.
23. Kaufman, N.A., Poznanski, R. and Guggenheim, K.: Eating habits of teenagers and self perception of body weight. Proceedings of the XXVI International Congress of Physiological Sciences, Jerusalem, 1974. Abstract.
24. Keen, H.: Incomplete story of obesity and diabetes. Keynote Address, First International Congress on Obesity, London, 1974.
25. Kennedy, G.C.: The role of depot fat in the hypothalamic control of food intake in the rat. *Proc. R. Soc. Lond.*, B 140:578–592, 1953.
26. Knittle, J.L. and Hirsch, J.: Effect of early nutrition on the development of rat epididymal fat pads: cellularity and metabolism. *J. Clin. Invest.*, 47, 2091–2098, 1968.
27. Larkin, R.: How hard will hyperphagic rats work for food? Presented at Eastern Psychological Association Annual Meetings, New York, N.Y., 1971.

28. Maller, O.: The effect of hypothalamic and dietary obesity on taste preference in rats. *Life Sci.*, 3:1281–1291, 1964.
29. Mayer, J., Gross, W., and Walker, J.W.: Taste and taste selection of food in hypoglycemia. *Brit. J. Exp. Pathol.*, 27: 297–305, 1946.
30. Moore, M.E., Linker, E. and Purcell, M.: Taste sensitivity after eating: A signal detection approach. *Am. J. Psychol.*, 78:107–111, 1965.
31. Moskowitz, H., Kluter, R.A., Westerling, J. and Jacobs, H.L.: Sugar sweetness and pleasantness: Evidence for different psychological laws. *Science*, 184:583–585, 1974.
32. Nachman, M.: Hypothalamic hyperphagia, finickiness and taste preferences in rats. Proceedings of the 75th Annual Convention of the American Psychological Association. 2:127–128, 1967.
33. Nisbett, R.E.: Taste, deprivation and weight determinants of eating behavior. *J. Pers. Soc. Psychol.*, 10:107–116, 1968.
34. Nisbett, R.E.: Determinants of food intake in human obesity. *Science*, 159:1254–1255, 1968.
35. Nisbett, R.E. and Kanouse, D.E.: Obesity, food deprivation and supermarket shopping behavior. *J. Pers. Soc. Psychol.*, 12:289–294, 1969.
36. Nordsiek, F.W.: The sweet tooth. *Am. Sci.*, 60:41–45, 1972.
37. Pangborn, R.M. and Simone, M.: Body size and sweetness preference. *J. Am. Diet. Assoc.*, 34:924–928, 1958.
38. Pfaffmann, C. and Hagstrom, E.C.: Factors influencing taste sensitivity to sugar. *Am. J. Physiol.*, 183:651, 1955.
39. Pfaffmann, C.: The Sensory and Motivating Properties of the Sense of Taste. In Jones, M.R. (Ed.), *Nebraska Symposium on Motivation,* University of Nebraska Press, 1961, pp. 71–110.
40. Price, J.M. and Grinker, J.: The effects of degree of obesity, food deprivation and palatability on eating behavior of humans. *J. Comp. Physiol. Psychol.*, 85:173–193, 1973.
41. Roswell, V.A. and Grinker, J.A.: Palatability and intake in two obese animal models: Genetically obese rats and ventromedial hypothalamic lesioned rats. Presented at the Eastern Psychological Association, Philadelphia, PA, 1974.
42. Schachter, S., Goldman, R. and Gordon, A.: Effects of fear, food deprivation and obesity on eating. *J. Pers. Soc. Psychol.*, 10:91–97, 1968.
43. Schachter, S.: Some extraordinary facts about obese humans and rats. *Am. Psychol.*, 26:129–144, 1971.
44. Sclafani, A.J. and Grossman, S.P.: Reactivity of hyperphagic and normal rats to quinine and electric shock. *J. Comp. Physiol. Psychol.*, 74:157–166, 1971.
45. Sclafani, A. and Springer, D.: Presented at Fifth International Conference on the Physiology of Food and Fluid Intake, Jerusalem, 1974.
46. Singh, D.: Effects of preoperative training on food-motivated behavior of hypothalamic hyperphagic rats. *J. Comp. Physiol. Psychol.*, 84:47–52, 1973.
47. Singh, D.: Role of preoperative experience on reaction to quinine taste in hypothalamic hyperphagic rats. *J. Comp. Physiol. Psychol.*, 186: 674–678, 1974.
48. Spiegel, T.A.: Caloric regulation of food intake in man. *J. Comp. Physiol. Psychol.*, 84:24–37, 1973.
49. Stern, J.S. and Greenwood, M.R.C.: A review of development of adipose cellularity in man and animals. *Federation Proc.*, 33:1952–1955, 1974.
50. Stunkard, A. and Koch, C.: The interpretation of gastric motility. *Archives of General Psychiatry*, 11:74–89, 1964.
51. Wampler, R.S.: Increased motivation in rats with ventromedial hypothalamic lesions. *J. Comp. Physiol. Psychol.*, 84:275–285, 1973.
52. Wooley, S.C.: Physiological versus cognitive factors in short-term food regulation in the obese and non-obese. *Psychosom. Med.*, 34:62–68, 1972.
53. Wooley, O.W., Wooley, S.C. and Dunham, R.B.: Can calories be perceived and do they affect hunger in obese and nonobese humans? *J. Comp. Physiol. Psychol.* 80:250–285, 1972.

54. Yensen, R.: Some factors affecting taste sensitivity in man: Food intake and time of day. *Quart. J. Exp. Psychol.*, 11:221–229, 1959.

DISCUSSION

JEROME: I have a very basic question; how are the moderately obese and the extreme obese defined?

GRINKER: In the Rockefeller Hospital, we typically see extremely obese individuals who are referred for treatment. They range in percent overweight (based on the Metropolitan Life Insurance tables) from 50 to 200% overweight, with a mean of approximately 100% (See Table 22–II). Moderately overweight individuals are arbitrarily defined as being from 25 to 50% overweight.

JEROME: Does this mean that anthropometric measurements are limited to weight determinations and that skinfold measurements are not done?

GRINKER: We determine total body water by tritiated water dilution and thus obtain lean body mass and total body fat. We therefore have a more reliable measure than skinfold measures.

MOSKOWITZ: I think, if I remember correctly, you may have a very important result in the preference judgments for the reduced obese. There is a reestablishment of the normal preference function for normal weight, previously obese people.

The "interpretation" problem shows up in a monotonic decrease (the misinterpretation via confusion of intensity and hedonics) and then you have a peak. The peak is a little lower. You still have a peak, rather than a monotonic decrease.

But that is not the important thing. There is a breakpoint. There is a concentration which they seem to like more than either lower or higher concentrations. It may not be statistically significant, but there is a breakpoint which was not there prior to the reduction. The exact location of the point is open to experimental investigation.

STELLAR: We find this in our normal weight subjects in that kind of experiment.

MOSKOWITZ: I imagine, too, that were you to use something like glucose instead of sucrose, the breakpoint would be a lot more exaggerated.

Sucrose typically gives a very smooth curve, which goes up and then smoothly goes down; whereas glucose goes sharply up and sharply down.

GRINKER: It is possible that our obese subjects were unable to separate judgements of intensity from judgements of pleasantness. This would explain the monotonic function that we got—as intensity increased, aversion increased. The slight peak that we get following weight reduction may occur because subjects no longer identify sweetness with calories and judge both as undesirable. Or the change may reflect real metabolic differences which occur when large changes in body weight or diet are made. We think that this breakpoint may be there based on our data from obese adolescents after weight reduction.

ENGEN: Is it likely that obese people

deliberately assign lower sweetness ratings because they do not want to be known as hedonists?

GRINKER: This was a major concern; whether or not we could believe our results, whether the sucrose aversion was a meaningful phenomenon. Subjects may have equated calories with sweetness and either denied any existing preference, or they may as Moskowitz suggests, have misinterpreted the task. This was a primary reason for looking at the actual consumption of these sucrose solutions and for looking at taste preferences in obese animals. Now, as I outlined very briefly, we are looking at taste parameters in children.

ENGEN: Do you tend to get more consistent results with animals?

GRINKER: Our human results are consistent, if you mean replicable. The question is what they mean. Our animal results are also consistent and certainly tend to support our findings. We are not getting the kind of increased intake in obese animals to make us question our human findings. We have now tested almost 100 obese humans and the sucrose aversion is clearly the predominant response pattern.

THOMPSON: Dr. Grinker's data indicating a difference between overweight and normal weight subjects in the hedonic ratings they assign to supra threshold concentrations of sucrose are intriguing. It may be helpful to examine more closely interindividual differences within each weight group to see if the pattern of hedonic ratings correlates with age of onset or degree of obesity.

My data suggest that there are at least two different types of hedonic responses to increases in sucrose concentration. Magnitude estimations of the intensity and pleasantness of sucrose concentrations ranging from 60 to 2000 mM were made by 16 normal and 24 overweight subjects after an overnight fast. There were 13 females and 3 males in the normal weight group and 17 females and 7 males in the overweight group. Sixteen subjects were 15% to 50% and 8 subjects were 50% to 149% above ideal body weight based upon the Metropolitan Life Insurance Tables.

All but one of the 16 normal weight subjects and 14 out of 24 overweight subjects gave their highest ratings of pleasantness to sucrose concentrations below 700 mM (Type I response). The rest of the subjects rated concentrations of sucrose greater than 700 mM as being most pleasant (Type II response). While there was no difference in age or sex between overweight subjects giving Type I and Type II responses, Type II responses were given by 50% of the moderately obese subjects but only 25% of the markedly overweight subjects. The reason for this difference is not readily apparent nor is it clear why one normal weight subject gave a Type II response.

STELLAR: Dean, are you asking them, in this situation, just to taste and rate pleasantness or are they under the threat of having to ingest what they taste?

THOMPSON: They simply taste the solution and expectorate.

STELLAR: I think it makes a great deal of difference, whether the subject has to face the consequence of ingesting what he tastes. When he rates it, especially after he has ingested it, he may rate something that your subjects rated pleasant, as un-

pleasant for complex post ingestional, rather than taste, reasons.

THOMPSON: Your comment serves as an introduction to a second part of the experiment. The effect of food ingestion on the type of hedonic response was tested by giving the subjects a 200 calorie Nutri-1000 liquid diet 30 to 60 minutes before the taste test. The meal produced no change in the pleasantness function for normal weight or for overweight subjects that gave Type I responses prior to eating. However, those obese subjects that gave Type II responses in the fasted state now found somewhat lower concentrations of sucrose to be the most pleasant. However, the inverted U-shaped curve generated when their magnitude estimates of pleasantness are plotted as a function of sucrose concentration still has its peak at a higher concentration than that observed for other subjects. Essentially the same results are obtained when the magnitude estimates of pleasantness are plotted as a function of magnitude estimates of intensity rather than as a function of sucrose concentration.

In conclusion, body state (hungry versus sated) affects the pleasantness judgements of overweight subjects that make Type II responses in the fasting state but not of overweight subject that respond in the manner characteristic of normal weight subjects. There are some, as yet, unexplained differences in the frequency of Type I and Type II responses among moderately and markedly obese subjects. If these findings can be replicated, their significance in relation to body weight regulation and caloric balance should be assessed in long term follow-up (ontogenic study) of adult as well as juvenile subjects for whom correlative weight histories are available. One might also look for changes in taste responses in the circumstance of weight loss or hormonal and metabolic variation.

LE MAGNEN: Have you examined and differentiated responses in juvenile onset and maturity onset obesities?

THOMPSON: The age range in these studies was from 18 to 60. We have not looked specifically at juvenile onset obesity.

GRINKER: Our subjects were of both juvenile and adult onset. We did not get any differences in sucrose detection or preference between juvenile and adult onset obese. There is a possibility that age at onset could differentiate taste responses since there is a tendency for the more overweight individuals to be of juvenile onset and there is a difference in taste preferences between the moderately overweight and the extremely obese. Additionally, there is a correlation between sucrose aversion and cellularity; the greater the cell number the greater the aversion.

Chapter 23

NUTRIENT FACTORS AS DETERMINANTS OF SUCROSE INGESTION

George Collier and Edward Hirsch

The caged, sedentary white rat, deprived of spouse, home, family, and frequently of food or water, has served as the model for most studies of ingestion. The sugar intake of such a nullity has been used to study taste receptors, preference, palatability, hedonics, and reinforcement. Traditionally, the rat has been viewed as a detector or measuring instrument rather than as an intact organism shaped by evolution to solve the problems of survival in a capricious environment. In the present chapter we speculate about the types of nutritional problems which the rat, a prototypic omnivore, would face in his natural environment under less contrived circumstances. More importantly, we use the ecological perspective of these speculations to interpret data on sucrose taste and intake generated within the conventional framework.

The omnivore's food does not come prepackaged in a nutritionally balanced form. It varies both in caloric density and in nutritional quality and composition. Yet the rat must select a diet that is both adequate and balanced from these diverse foods sources. He must compensate for the deficiencies or excesses of preceding meal(s) over relatively short periods of time. Since omnivores must detect and eat food that varies widely in nutritional composition while maintaining appropriate levels of energy intake and a nutritionally balanced diet, they should be "experts" in both dietary self-selection and caloric adjustment. The omnivore's task contrasts sharply with that of either the carnivore or herbivore. The herbivore is usually surrounded by easily available foods of low nutritional quality that are processed to meet his energetic and nutritional needs by means of his complex digestive system. The carnivore, on the other hand, exploits foods of low availability but of high nutritional quality. Both groups confront foods that are relatively constant in caloric density and, therefore, they can use bulk to gauge calories.

In contrast to his laboratory compatriot, our freely wandering, hypothetical rat faces a world that is not as benevolent as that arranged by the typical researcher. Food must be

Acknowledgment: This research was supported by Research Grants HD 00941 and HC 03279 from the National Institutes of Health, Bethesda, Maryland, and from funds from the Rutgers University Research Council. Some of the data reported were gathered by the second author while he was an NAS-NRC Visiting Scientist at the U. S. Army Natick Laboratories. The authors wish to thank Carolyn Kent Rovee of Douglass College, Rutgers University, for her critical reading of the manuscript.

sought or procured, an obviously more energetic process than bending over a food cup and ingesting a "balanced" diet. It seems likely that under all but the most idyllic circumstances, food-gathering accounts for a substantial portion of the rat's energy budget. Information on this cost is lacking, but its existence inspires the conjecture that the optimal strategy for our hypothetical rat would entail anticipating needs rather than responding to them. In this manner, deprivation or, even worse, depletion, with their attendant uncertainties, would be precluded. This strategy would ensure an adequate supply of energy for the long and sometimes fruitless search for sustenance. The density of prey or vegetation and its availability in time and space should be major influences on feeding patterns. The character of the ecological niche the animal is exploiting may be a more important determinant of feeding patterns than the physiology of the animal. Unfortunately, or perhaps fortunately for these speculations, there are very few data from the field or laboratory on the freely feeding animal of the character that would reflect on these issues. Some data exists supporting this conjecture for carnivores [e.g., 10,11,18,19,31] and herbivores [2] but none for omnivores. In this chapter, we examine older laboratory data, gathered originally for other purposes, for insights into the mechanisms that have evolved to solve nutritional problems in the freely feeding omnivore under field conditions.

Our omnivorous rat's sugar consumption is viewed within the framework of his attempt to select a diet that is not only balanced but also supplies the appropriate number of calories. Sucrose, a pure and highly palatable carbohydrate, supplies one of the basic dietary components. Its consumption cannot be divorced from the intake of other dietary items. We will look first at sugar consumption in relation to body weight regulation and then from the perspective of dietary balance.

BODY-WEIGHT REGULATION

The now classic papers of Adolph [1] and Mayer [23] revealed that the rat is capable of regulating body weight under a wide variety of circumstances. Intake is adjusted to expenditure following extended bouts of exercise [24] exposure to the cold [12] or caloric dilution.[1] such results have been replicated by many researchers and are the backbone of most theories of the control of food intake. One widely held intuitive hypothesis is that growth rate and/or final weight should be influenced by dietary palatability. The asymptotic body weights should be higher on more palatable diets. But with the exception of data collected from brain-damaged animals [9,27,33,34] there is little evidence that intake or weight is influenced by dietary palatability in the long term. Thus, we ask whether providing the rat with access to sugar along with his usual fare will affect his body weight. One problem with experiments attempting to answer this question of the effect of dietary variables on the regulation of energy balance has been the failure to evaluate the results against a reference body weight ob-

tained on reference diet under standard conditions of temperature, cage size, exercise, level of disease, season, etc. Figures 23–1 and 2 present the results of two experiments. The rats were provided *ad lib* access to a drinking tube containing sucrose solution, another tube containing water, and a cup of Purina chow. In the second experiment consummatory effort was varied by means of the number of licks required to obtain a given volume of fluid. The top two panels of each figure show that total caloric intake and body weight level do not differ across groups. There is a trend, however, for those rats offered the most concentrated sugar solution to be heavier. This trend might have attained significance if the experiment had been longer than 40 days.[26]* The point of interest, with this small caveat, is that rats regulate body weight within narrow limits when offered a highly palatable supplement to their normal diet.

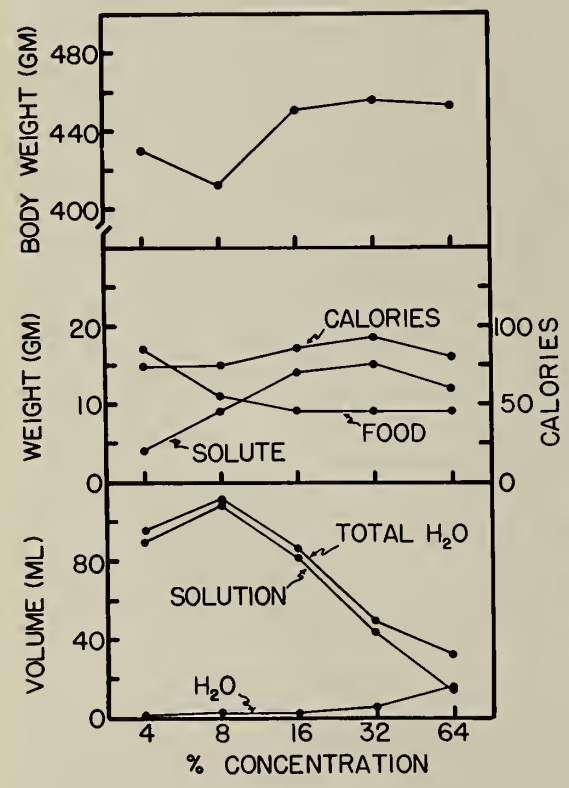


Figure 23–1. Intake parameters of sucrose ingestion as a function of sucrose concentration.

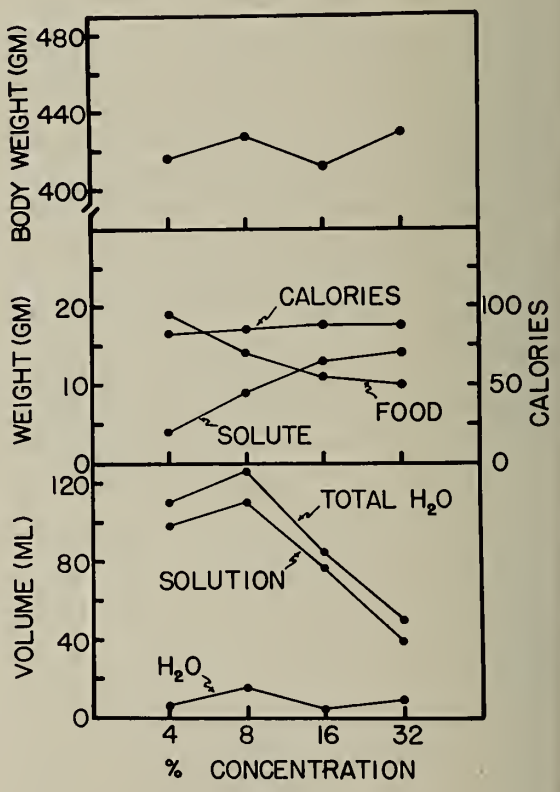


Figure 23–2. Intake parameters of sucrose ingestion as a function of sucrose concentration.

* Hirsch, E. Priorities and compromises in the defense of body weight. Symposium paper presented at the 46th annual meeting of the Eastern Psychological Association, New York, April 3, 1975.

REGULATION OF DIET COMPOSITION

Our hypothetical omnivore does not have access to the "optimum" diet constructed by nutritional experts working for rat food suppliers. He picks and chooses among a wide variety of items differing in taste, texture, and many nutritional aspects. A long series of selection experiments [20] has demonstrated the rat's capacity to select among such an array of items to produce a diet which will provide for normal growth and maintenance of body weight. Most doubts about this capacity have arisen in experiments using purified components.[7]

Rather than asking the rat what items he will choose, one may ask which he prefers. The so-called preference experiments that stem from the latter question most often look at the relative intakes of a single item varying in concentration. To consider only one component at a time, however, is misleading. In fact, one must consider the total food economy of an animal in order to understand the intake of any single component.

Sugar intake

In the prototypical experiment on animal taste preference the rat is offered one of a number of sugar solutions that differ in concentration. Typically, volume drunk is the index of preference.[28] The results of one such experiment are shown in the bottom panels of Figures 23–1 and 2. Sucrose is available for a 24-hr time period. Peak intake of solution occurs at the 8% concentration. But is this the "most-preferred" solution? Is volume drunk the most appropriate measurement for conferring the title of "most preferred"? Several lines of evidence suggest that volume ingested reflects the influence of controlling processes other than preference per se. McCleary,[25] in his now classic paper, suggests that at least two factors, taste and postingestive consequences, must be considered in accounting for the intake of sapid solutions (see also Stellar [32]). The multifactor approach advocated by Adolph [1] and McCleary [25] suggests two alternative analyses of the preceding "preference" experiment. Sucrose intake can be examined in relation to the intake of the maintenance diet (Purina chow) and of water. The middle panels of Figures 23–1 and 2 show that solute consumed is an increasing function of concentration up to 32%. Food intake falls as solute intake increases. The total caloric intake and body weight remain relatively constant across concentrations. If solute intake is taken as the measure of preference, the 32% solution would gain the title of "most preferred."

An examination of the proportion of the total caloric intake obtained from the sugar solutions at each concentration suggests that none of the solutions are the "most preferred." Figure 23–3 shows that over the range of concentrations from 16% to 64%, the proportion of calories from sugar remains constant at 60%. This finding, which has been verified in a number of subsequent experiments under a wide variety of conditions, suggests that preference experiments are really "dilution" experiments. Adolph [1] and many subsequent researchers have shown that when a diet is diluted by a non-nutritive compo-

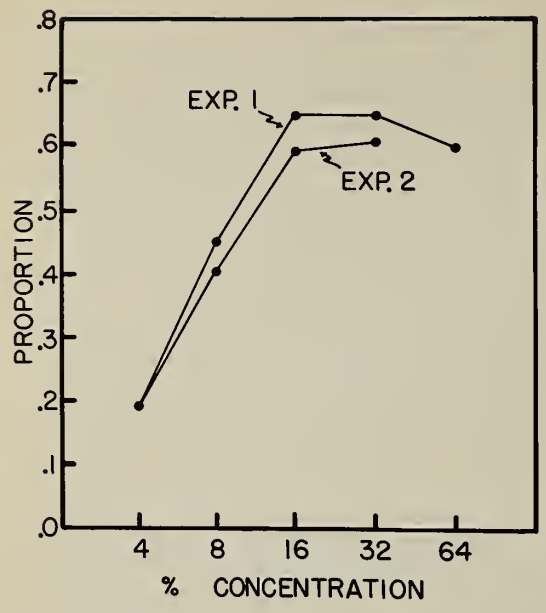


Figure 23–3. Proportion of total calories taken from sucrose.

nent such as celluflour or kaolin, rats maintain caloric intake through a compensatory increase in their intake of the more dilute diets. In the present experiment, one separately offered component of the diet is diluted by water. The rat appears to compensatorily increase his intake of the more dilute offerings in such a fashion that the total intake of the sugar component remains constant. This interpretation raises two questions: (a) What is the rat regulating?; and (b) Why does the rat fail to maintain solute intake on the most dilute (4% and 8%) solutions?

Some insight into what it is the rat is regulating can be gained from an examination of the intake of the various components. The sucrose component of the diet is a pure carbohydrate. The Purina chow component is a mixture of carbohydrates, fats, proteins, minerals, vitamins, and non-nutritive bulk. Purina chow contains about 23% protein by weight. Self-selection studies to be reported subsequently show that this is high relative to what an age-mate rat will freely choose if offered the dietary components separately.

The fact the Purina chow is a "high" protein diet from the rat's point of view has seldom been recognized in nutrition experiments. This level of protein may influence the intake of sucrose solutions. The rat is simply trying to adjust the proportions of carbohydrate and protein in his diet. This level of protein in commercial chow is in response to the fact that protein, in spite of added antioxidants, undergoes oxidation at room temperature when exposed to air. High initial levels thus ensure adequate levels of protein in an "old" bag of chow.

The ideal form of the selection experiment would have the "significant" dietary components offered in pure form. This is not difficult with carbohydrates or fats, but protein presents a problem. When purified proteins such as casein or Drackett are offered, rats tend either to not eat them at all or to reduce their intake and fail to show normal growth or maintenance of body weight. There are many possible reasons for the rat's aversion to purified proteins, but there are few systematic data. It seems to be necessary to present the protein fraction either in a form (unpurified) that is naturally diluted by other substances or to adulterate the purified form in order to obtain standard growth. When offered sucrose solutions and Purina chow, the rat characteristically reduces his protein intake to approximately 10%, a value which is close to the value a rat at his age will take in an

experiment in which a more concentrated protein and a carbohydrate are offered separately. Thus, we would like to sugest that the usual single-bottle preference study is, in reality, a self-selection study in which one of the diet components is being diluted. Optimum total caloric intake and optimum diet composition are both defended.

The failure of the rat to consume sufficient amounts of the most dilute sucrose solutions can be illuminated by calculating the amount of solution at these values the rat would have to consume in order to achieve 60% of his caloric intake from sucrose. These values are 350 ml for the 4% solution and 175 ml for the 8% solution, amounts simply too large for the rat to consume. This finding is consistent with Adolph's [1] chow dilution experiments in which rats were unable to compensate completely for diets more dilute than 40–50%.

The preceding analysis indicates that volume consumed does not necessarily reflect preference. Is choice behavior in a two-bottle situation a better index? The results of an experiment in which animals are offered a pair of sucrose solutions are shown in Table 23–I. The solutions were offered on an *ad lib* basis, 24 hr/day for 6 days at each pair.[4] The rats always drink more of the most concentrated solution of the pair. When the concentrations are the same, side preferences are exhibited. Table 23–II shows the proportion of the total caloric intake taken from the two solutions. These data are consistent with those obtained from the single-bottle tests. When either member of the pair of sucrose solutions was 16% or greater, the animals take approximately 60% of their total caloric intake from sucrose. This shows that at least two factors are operating simultaneously. The first factor determines choice between concentrations, and the second factor the total amount of solute consumed.

These experiments establish that

TABLE 23–I

VOLUMES CONSUMED FROM PAIRS OF SUCROSE SOLUTIONS

Concentration	*4%*	*8%*	*16%*	*32%*
4%	42/45	11/87	16/71	12/44
8%	...	35/52	37/81	22/48
16%	...	...	36/55	22/36
32%	...	...	...	15/46

Note. Entries show volume consumed of concentration indicated at left over volume of concentration indicated at top of table.

TABLE 23–II

PROPORTION OF TOTAL CALORIC INTAKE TAKEN FROM PAIRS OF SUCROSE SOLUTIONS

Concentration	*4%*	*8%*	*16%*	*32%*
4%	.10	.36	.56	.58
8%	...	.23	.60	.62
16%	...	...	.60	.63
32%	...	...	...	.53

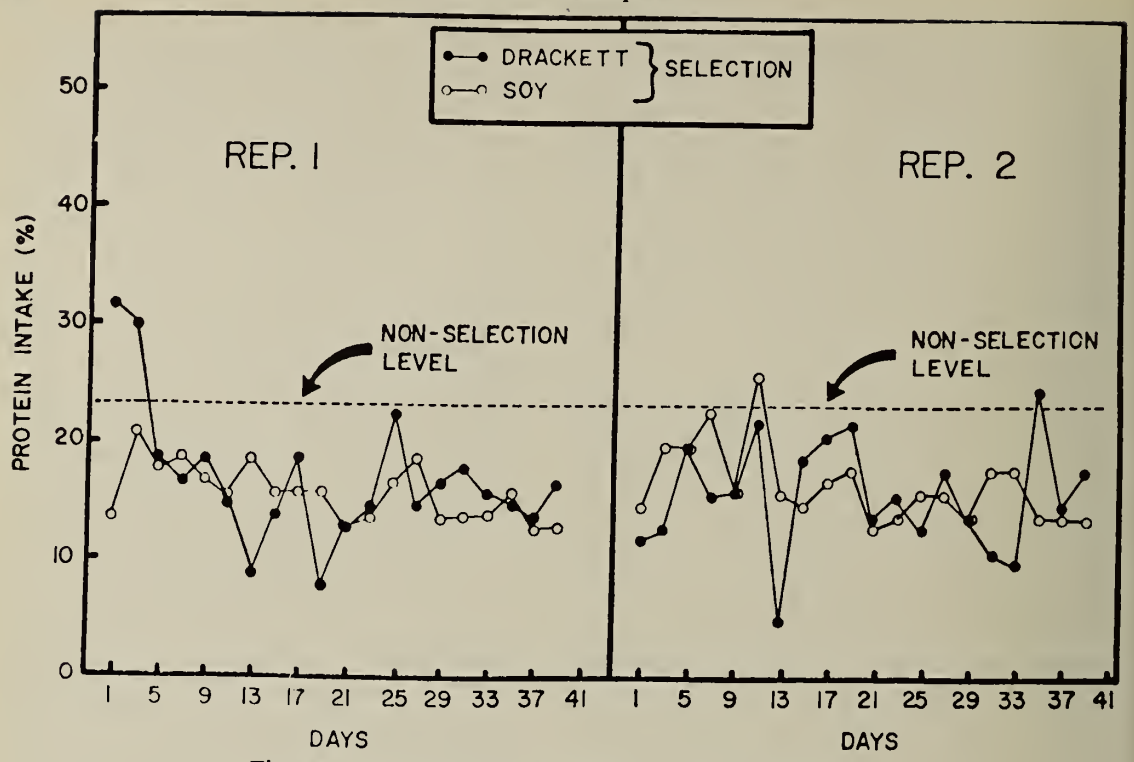


Figure 23–4. Daily percentage of diet as protein.

under certain conditions rats are able to control both caloric intake and diet composition. It becomes of interest to ask how this control varies when the character of the dietary components is manipulated or when the energy and protein requirements of the animal change due to differing environmental conditions or age. Unfortunately, it is not possible to report a systematic set of experiments, but some indications of the direction of the results can be gathered from the somewhat disjointed series of experiments we present.

Palatability and dilution of the protein and carbohydrate components

In the preceding experiments, increased intake compensated for dilution of the carbohydrate component at all but the most dilute concentrations. Rozin [29] reports similar results for the dilution of the protein component, although he failed to find compensation for dilution of the carbohydrate component of the diet.

Experiments using sugars which differ in sweetness show compensations similar to that obtained for sucrose.[e.g., 16] Unfortunately, most investigators who have used other sugars have failed to record the concurrent food intake, so that the influence of the kind of sugar on total caloric intake and the proportions of protein and carbohydrate consumed is not known. In a series of experiments using dextrinized starch, a relatively tasteless powder, as the source of carbohydrate and a soy protein as the source of protein, it was found that young rapidly growing rats constructed a diet containing 16% pro-

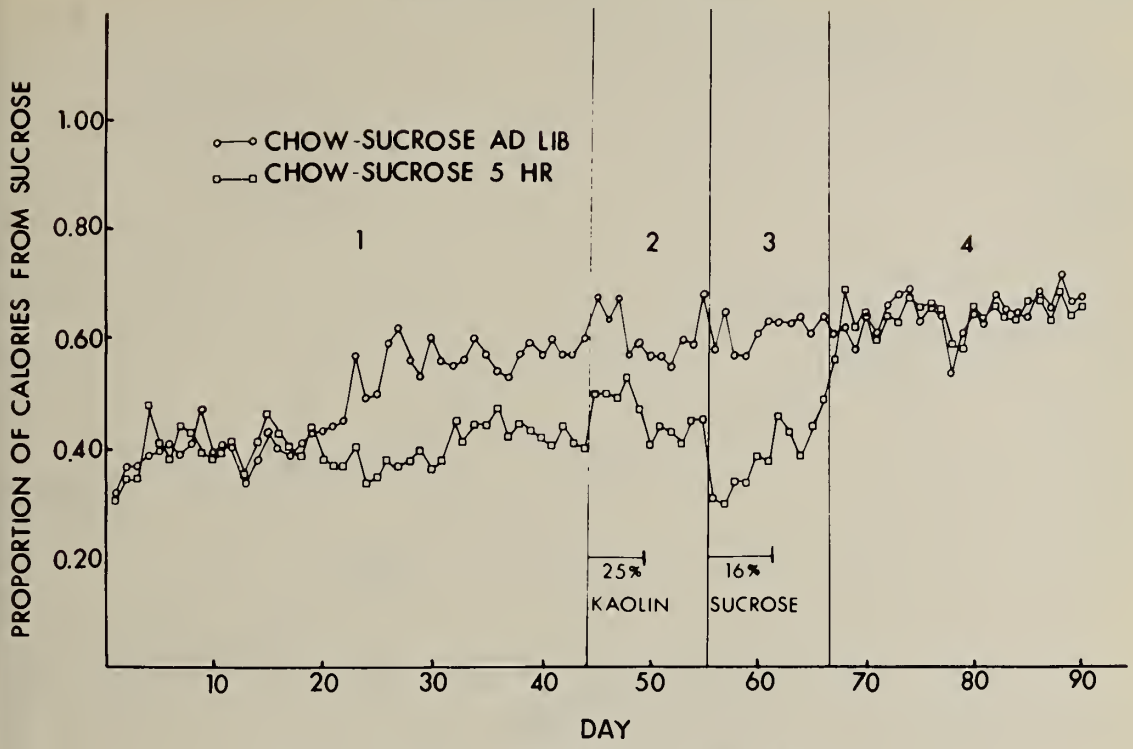


Figure 23–5. Daily percentage of diet as sucrose.

tein. This value is close to that obtained for sucrose,[7,8] and suggests that the role of sweetness in selection may be relatively minor.

There is, however, some evidence that dietary balance is more strictly defended than total caloric intake. Several investigators have reported that rats fed sugar along with a complete commercial chow grew faster than rats denied the sugar.[26] Although there is a small elevation of caloric intake, the proportion of protein to carbohydrate consumed is unaffected.

Again, there are few systematic experiments manipulating different sources of protein. One such experiment is available. Two protein sources were used in a self-selection experiment.[8] These proteins were derived from soybean oil meal (SBOM). One, Drackett (ADM Assay Protein C–1), was a purified SBOM containing 81% protein, and the other, a standard SBOM containing 46% protein. These two proteins were mixed with the other essential dietary components to provide a 23% protein diet. In the Drackett group, food intake and growth are depressed relative to the normal protein group even though both diets contained the same percent of protein. This finding has often been reported for casein (See ref. 20). When the protein and carbohydrate components are offered separately, a similar result is obtained; that is, there is a depression of the intake in the Drackett group and poorer growth. However, the percent protein consumed by both the regular SBOM and the Drackett groups remained the same, 16% (see Fig. 23–4). Thus, when the protein component is not palatable, it depresses the intake

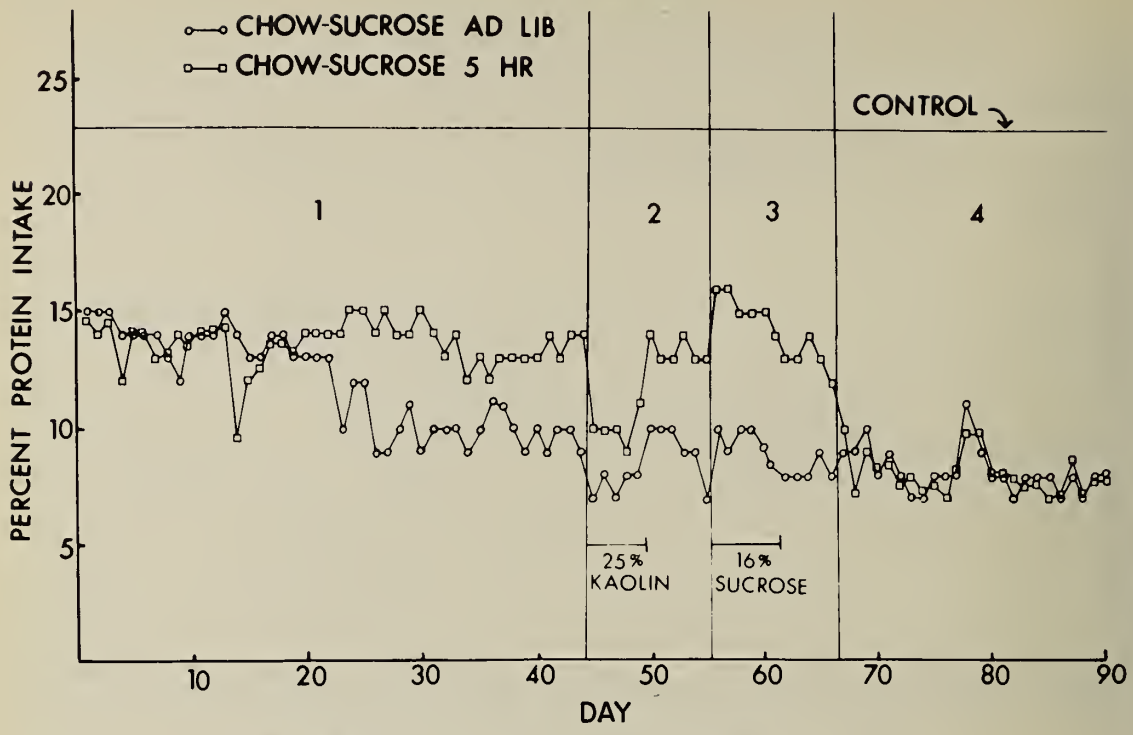


Figure 23–6. Daily percentage of diet as protein.

of *both* components, and percentage protein intake is defended. A palatability change which affects the intake of *either* component will result in an increase or decrease in intake of that component with a compensatory change in the other, conserving the ratio of protein to carbohydrate.

Age

The rat's protein requirement is markedly influenced by age and is at a maximum in the young, rapidly growing animal.[35] Using the well-documented observation that food deprivation potentiates the intake of palatable carbohydrate solutions,[5,17] an attempt was made to induce a distortion of the protein/carbohydrate ratio. Weanling rats were offered food for 5 hr/day and compared with age-mates having *ad lib* access to food. Within each food availability condition, one group of animals was allowed access to a 32% solution of sucrose in addition to a balanced diet. It was predicted that the restricted animals would over-eat from the palatable carbohydrate source and in this manner would distort the protein/carbohydrate ratio and lower the amount of protein ingested. Figure 23–5 shows that under these conditions the restricted animals take a lower rather than a higher proportion of their calories from the sucrose solution. They select a diet that is appropriate for their smaller body size. For both groups of animals, the percent protein intake declines with age (see Fig. 23–6). This level of protein intake is substantially lower than the control level (23% Purina chow) but is adequate for optimal growth (Fig. 23–7).

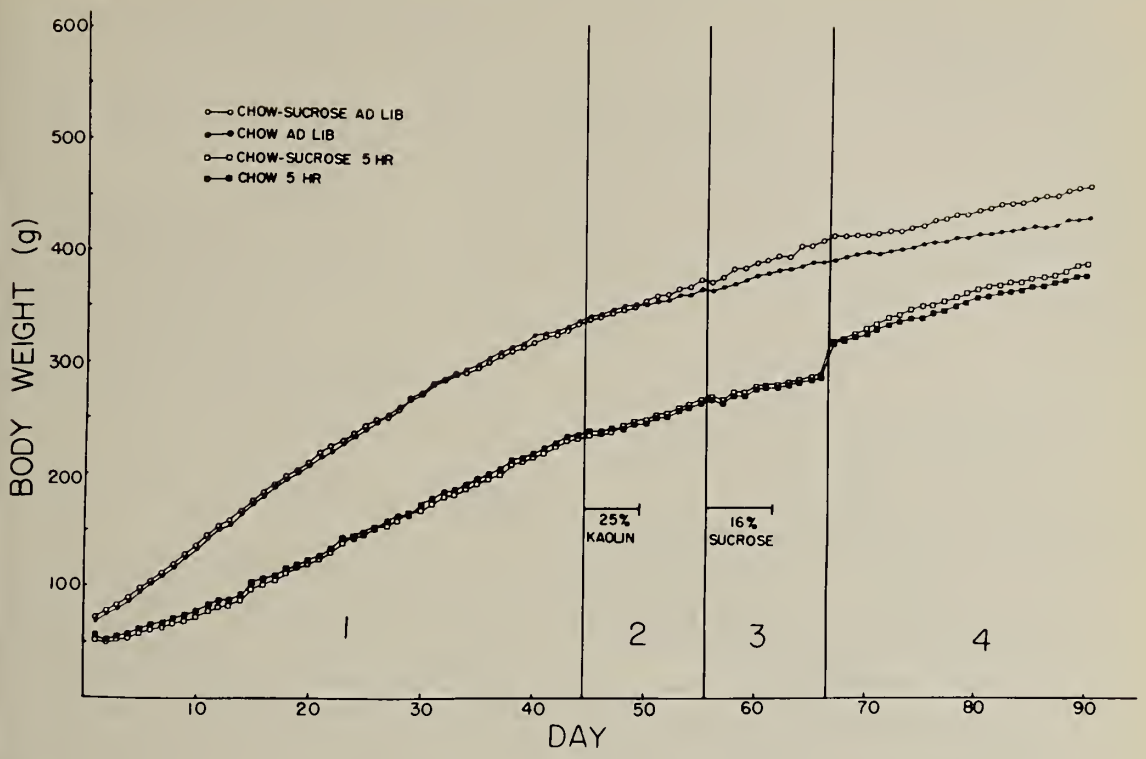


Figure 23–7. Body weight.

ENERGY BALANCE

Once the essential requirements for minerals, vitamins, and fats are met, the two limiting requirements for growth and maintenance are protein and energy. The sedentary laboratory rat raised under standard conditions of temperature, ventilation, and lighting reaches maximum growth rates with a diet (chow) of 16% protein. The question can be asked as to how the rat manages to meet the additional energy requirements posed by low temperatures, access to exercise, or deprivation of the normal ration.

Cold

Rats reared at 2°C increase their food intake by approximately 20%.[22] Even so, cold-reared animals grow less well than animals reared at room temperature. Self-selecting animals in both groups grow as well as nonselecting animals. In the selecting animals, the increase in food intake is accomplished by increased intake of carbohydrate while protein intake is unchanged. Thus, a lower percentage of the diet is protein (Fig. 23–8). These results show that animals (rats) allowed a choice will meet the additional energy requirements posed by cold by increasing their carbohydrate intake. While the physiology of this choice has not been explored, it seems likely that it relates to the relative costs of obtaining energy from carbohydrate and protein. Amino acids must be deaminated before they can enter the energy cycle. It is interesting

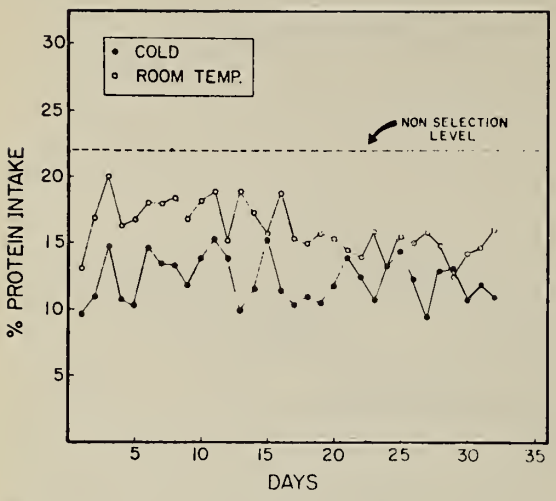


Figure 23–8. Daily percentage of diet as protein.

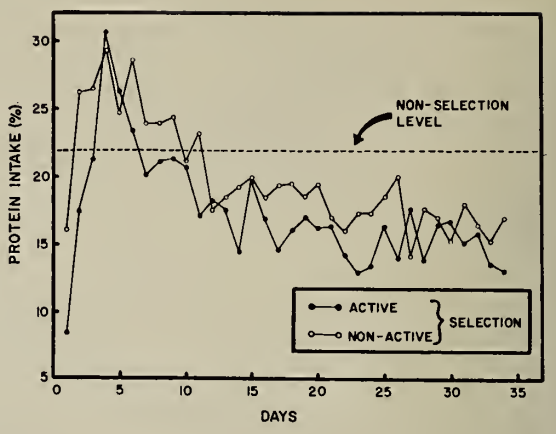


Figure 23–9. Daily percentage of diet as protein.

that these differences can be so sensitively reflected in choice behavior.

Activity

In growing animals, access to running wheels yields animals with body weights approximately 12% less than that of inactive controls.[7] The introduction of wheels leads to a transitory reduction in food intake with a return to normal intake within 5 to 10 days.[3] The difference in size is reflected in the amount of adipose tissue. The lean body mass does not differ.[21] In the selecting rats who had access to activity wheels, there was a small but significant (2%) reduction in the proportion of protein consumed and thus an increase in carbohydrate intake (see Fig. 23–9). This result again shows that animals free to select their own diet meet different energy requirements by changing the proportions of carbohydrate to protein. Figure 23–10 shows the results of a somewhat different activity-selection experiment.* Rats are offered protein and three sources of energy: dextrinized starch, sucrose and corn oil. There is an active (running wheels), less active (double cages), and inactive (regular cages) group. It can be seen that the proportion of the total caloric intake from some of the dietary items is a function of activity. Carbohydrate and fat are affected by activity, but protein was not. The most striking result was a shift from a preference for dextrinized starch in the inactive animals to a preference for sucrose in the active animals. The shift is accompanied by a drop in fat intake in the active group. This result is, again, a strong indicator that the state of energy balance has an important effect on self-selection and that one cannot evaluate diet selection without considering environmental conditions.

Deprivation

What is the effect of restricted intake on the selection of protein and carbohydrate? In an experiment in which total *ad lib* caloric intake of 64 calories was reduced to 43 calories (resulting in a 10% weight loss), the

* Sunday, S. Dietary self-selection and activity levels in the developing rat. Paper presented at the 46th annual meeting of the Eastern Psychological Association, New York, April 3, 1975.

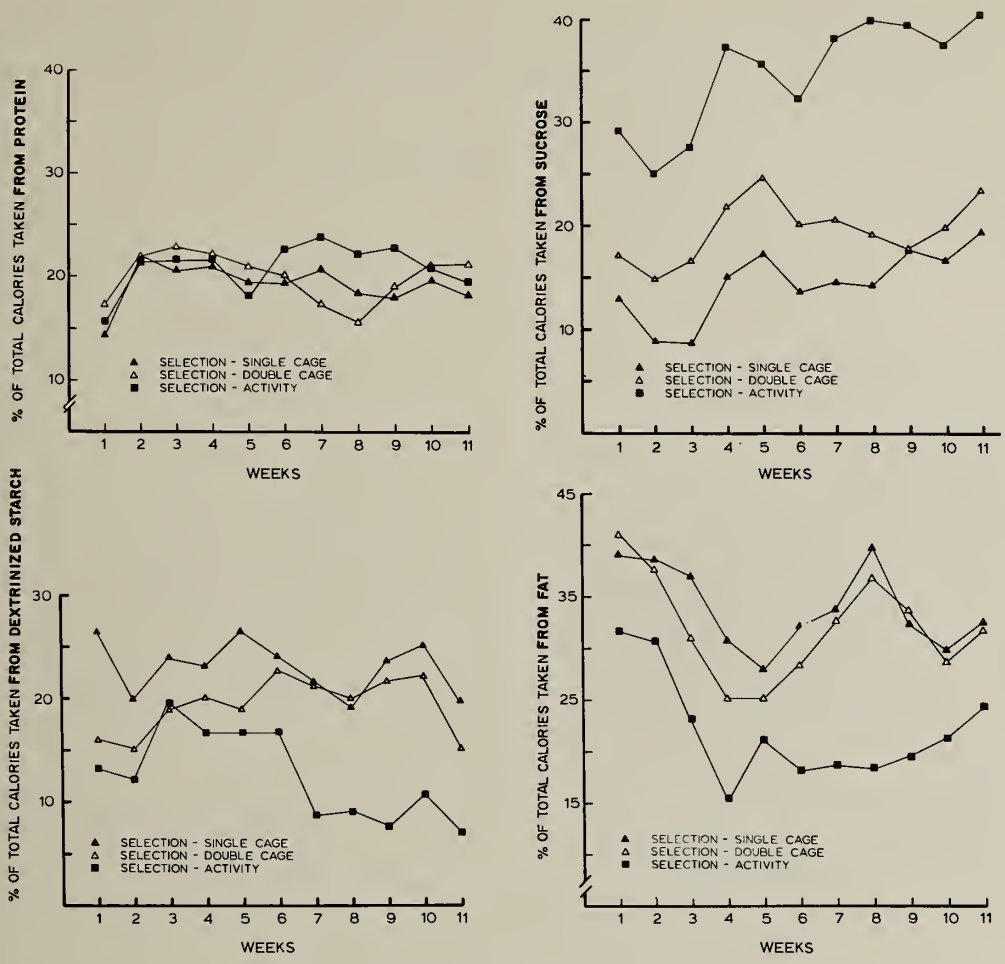

% OF TOTAL CALORIES TAKEN FROM PROTEIN
% OF TOTAL CALORIES TAKEN FROM SUCROSE
% OF TOTAL CALORIES TAKEN FROM DEXTRINIZED STARCH
% OF TOTAL CALORIES TAKEN FROM FAT
SELECTION - SINGLE CAGE
SELECTION - DOUBLE CAGE
SELECTION - ACTIVITY
WEEKS

Figure 23–10. Daily percentage of diet as protein, sucrose, dextrinized starch and fat.

proportion of calories taken from sucrose in a limited session is higher than that taken when food and sucrose are given *ad lib.*[5] Following this lead, Navarick* offered rats food (Purina chow) on a 1 hr/day schedule. Animals initially lose weight on this schedule. When sucrose is available, the amount of sucrose consumed continuously increases as a function of weight loss, and the rats eventually die. Control animals offered only Purina chow adapt to the schedule over time and eventually recover their normal body weight. These results are replicated by Hamilton [13] and Sclafani [30] and show that starvation-induced increases in an animal's energy requirements leads to higher intake of carbohydrate relative to protein. In the wild this tactic provides energy for searching for food and for the maintenance of metabolism, but in the laboratory, it is fatal. These results stand in sharp contrast to the data previously described in which weanling rats were limited to 5-hr ac-

* Navarick, D. J. Effect of saccharin and sucrose consumption on adjustment to a food deprivation schedule. Unpublished master's thesis, Rutgers University, 1969.

cess to chow and sucrose. When 5-hr access is allowed, optimal selection occurs, but with only a 1-hr period of access, excess carbohydrate intake prevails. This may be the result of the stronger protein requirement in rapidly growing rats, or the degree of deprivation.

Availability

In the natural feeding situation where an animal is free to initiate and terminate a meal independently of the experimental design of the researcher, the effort which the animal must expend to get a meal becomes an important parameter of meal-taking. A series of papers [6,15,18] demonstrates that as availability of food decreases, animals adopt the tactic of reducing the number of meals they initiate and increasing the duration of each meal. Total intake is conserved.

Again, in the natural feeding situation, it seems likely that the items necessary to construct a balanced diet may vary in their availability, presenting the omnivore with the problem of choosing the easily available item and imbalancing his diet, or of doing the work to obtain the required item and maintaining a balanced diet. This environment can be simulated in the laboratory by offering sucrose *ad lib* and requiring rats to lever-press on increasing fixed-ratio schedules for a balanced diet (Noyes pellets). *Ad lib* rats, like those that perform only a single bar press to obtain each pellet, take 60% of their caloric intake from sucrose. As the size of the ratio increases, the percent of calories taken from sucrose increases and the amount of protein obtained from the Purina chow decreases gradually. The amount of protein taken never fell below 4–5% of the diet. The rats expend up to 40,000–50,000 responses per day (16 hr of bar-pressing) in order to obtain Noyes pellets, the protein-containing fraction. This level of protein intake is sufficient to sustain weight gain in these older animals.* The same results are obtained when Purina chow was offered *ad lib,* but the animal has to work for sucrose. Thus rats are willing to perform a large amount of instrumental behavior to maintain the "preferred" ratio of protein to carbohydrate in their diet.† It should be pointed out that these rats are not deprived; that is, they obtained all of their food in the situation and maintained normal caloric intake. These results show that the maintenance of a balanced diet has powerful motivating effects. Rats will expend large amounts of effort to obtain a balanced diet.

IMPLICATIONS FOR CARBOHYDRATE INTAKE

It is clear from the results presented in this chapter that the intake of carbohydrate, or more specifically, sucrose, cannot be viewed outside of the framework of the total nutritional economy of the animals being studied. Rather, carbohydrate intake must be viewed in the context of the concurrent intake of other nutrients, the composition of these nutrients, the relative palatability of the components, the state of energy balance, and the availability of items of diet. The omnivorous rat has evolved

* Hirsch, E. Unpublished data.
† Collier, G. Unpublished data.

mechanisms, as yet little understood, which enable him under control circumstances to select a nutritionally balanced diet. Excessive or deficient intakes must result from aberrant environments or physiological abnormalities.

This analysis raises additional questions concerning the niches occupied by animals. It seems likely that the problems facing herbivores and carnivores are very different from those confronting the omnivore. The generalized herbivore eats a diet notorious for its poor quality both in calories and in nutrition. However, he processes his intake in a complex digestive tract which extracts or manufactures the requisite nutrients. It appears that he is capable of regulating caloric intake by adjusting *feed efficiency.*[14] Thus it seems likely that he would be less sensitive to either the caloric or the nutritional dimensions of the diet. Studies by Hirsch [14] show that the guinea pig, a monogastric herbivore, is surprisingly insensitive to the caloric dilution of his diet and appears to regulate intake by bulk. The necessary nutritional balance studies have not been performed.

The carnivore, on the other hand, eats a food which replicates his own composition almost exactly and which is calorically dense. The carnivore should exhibit little talent for selection or caloric regulation. Preliminary studies on house cats [18] suggest that they tend to eat for bulk, losing weight on diluted diets and gaining weight on concentrated ones. Whether carnivores are capable of dietary selection remains an unanswered but intriguing question.

These data and the accompanying speculations suggest that one cannot understand either the pattern of intake or the relative amounts consumed of the available food items without considering the total nutritional economy of the animal and the ecological niche which he exploits.

REFERENCES

1. Adolph, E. F.: Urges to eat and drink in rats. *Amer. J. Physiol.* 151: 110—125, 1947.
2. Bell, R. H. V.: A grazing ecosystem in the Serengeti. *Scient. Am.* 224: 86—93, 1971.
3. Collier, G.: Work: A weak reinforcer. *Trans. N. Y. Acad. Sci.* (Series II) 32: 557—576, 1970.
4. Collier, G. and Bolles, R.: Some determinants of intake of sucrose solutions. *J. comp. physiol. Psychol.* 65: 379—383, 1968.
5. Collier, G. and Bolles, R.: Hunger, thirst and their interaction as determinants of sucrose consumption. *J. comp. physiol. Psychol.* 66: 633—641, 1968.
6. Collier, G., Hirsch, E. and Hamlin, P.: The ecological determinants of reinforcement in the rat. *Pysiol. & Behav.* 9: 705—716, 1972.
7. Collier, G., Leshner A.I., and Squibb, R.L.: Dietary self-selection in active and nonactive rats. *Physiol. & Behav.* 4: 79—82, 1969.
8. Collier, G., Leshner, A.I. and Squibb, R.L.: Self-selection of natural and purified dietary protein. *Physiol. & Behav.* 4: 83—86, 1969.
9. Corbit, J. and Stellar, E.: Palatability, food intake and obesity in normal and hyperphagic rats. *J. comp. physiol. Psychol.* 58: 63—67, 1964.
10. Estes, R. D.: Predators and scavengers. Part I. *Nat. Hist. N. Y.* 76: 20—29, 1967.
11. Estes, R.D.: Predators and scavengers. Part II. *Nat. Hist. N. Y.* 76: 38—47, 1967.

12. Hamilton, C. L.: Food and temperature. In Code, C.F. (Ed.) *Handbook of Physiology. Vol. 1: Alimentary Canal,* Washington, American Physiological Society, 1967, pp. 367—386.
13. Hamilton, L. W.: Starvation induced by sucrose ingestion in the rat: Partial protection by septal lesions. *J. comp. physiol. Psychol.* 77: 59—69, 1971.
14. Hirsch, E.: Some determinants of intake and patterns of feeding in the guinea pig. *Physiol. & Behav.* 11: 687—704, 1973.
15. Hirsch, E. and Collier, G.: The ecological determinants of reinforcement in the guinea pig. *Physiol. & Behav.* 12: 239—249, 1974.
16. Jacobs, H. L.: Some physical, metabolic and sensory components in the appetite for glucose. *Amer. J. Physiol.* 203: 1043—1054, 1962.
17. Jacobs, H. L. and Sharma, K. N.: Taste versus calories: Sensory and metabolic signals in the control of food intake. *Ann. N. Y. Acad. Sci.* 157: 1084—1125, 1969.
18. Kanarek, R. B.: Availability and caloric density of the diet as determinants of meal patterns in cats. *Physiol. & Behav.,* 15: 611–618, 1975.
19. Kruk, H.: *The Spotted Hyena: A Study of Predation and Social Behavior.* Chicago, University of Chicago Press, 1972.
20. Làt, J.: Self-selection of dietary components. In code, C.F. (Ed) *Handbook of Physiology. Vol. 1: Alimentary Canal,* Washington, American Physiological Society, 1967, pp. 367–386.
21. Leshner, A.I.: The adrenals and the regulatory nature of running wheel activity. *Physiol. & Behav.* 6: 551–558, 1971.
22. Leshner, A.I., Collier, G., and Squibb, R.L.,: Dietary self-selection at cold temperatures. *Physiol. & Behav.* 6: 1–3, 1971.
23. Mayer, J.: Regulation of energy intake and the body weight: The glucostatic theory and the lipostatic hypothesis. *Ann. N.Y. Acad. Sci.* 63: 15–43, 1955.
24. Mayer, J., Marshall, N. B., Vitale, J.J., Christiansen, J.H. Masayekhi, M. B. and Stare, F.J.: Exercise, food intake and body weight in normal rats and genetically obese adult mice. *Amer. J. Physiol.* 177: 544–548, 1955.
25. McCleary, R. A.: Taste and post-ingestive factors in specific hunger behavior. *J. comp. physiol. Psychol.* 46: 411–421, 1953.
26. Muto, S. and Miyahara, C.: Eating behavior of young rats: Experiment on selective feeding on diet and sugar solution. *Br. J. Nutr.* 28: 327–337, 1972.
27. Panksepp, J.: A reexamination of the role of the ventral medial hypothalamus in feeding behavior. *Physiol. & Behav.* 7: 385–394, 1971.
28. Pfaffmann, C.: The pleasures of sensation. *Psychol. Rev.* 4: 253–268, 1960.
29. Rozin, P.: Are carbohydrate and protein intakes separately regulated? *J. comp. physiol. Psychol.* 65: 23–29, 1968.
30. Sclafani, A.: Feeding inhibition and death produced by glucose ingestion in the rat. *Physiol. & Behav.* 11: 595–602, 1973.
31. Schaller, G.B.: *The Serengeti Lion: A Study of Predator-Prey Relations.* Chicago, University of Chicago Press, 1972.
32. Stellar, E.: Drive and motivation. In Field, J. (Ed). *Handbook of Physiology. Vol. 3: Neurophysiology,* Washington, American Physiological Society, 1960, pp. 1501–1527.
33. Teitelbaum, P.: Motivation and control of food intake. In Code, C. F. (Ed). *Handbook of Physiology. Vol. 1: Alimentary Canal,* Washington, American Physiological Society, 1967, pp. 319–335.
34. Teitelbaum, P. and Epstein, A. N.: The lateral hypothalamic syndrome: Recovery of feeding and drinking after lateral hypothalamic lesions. *Psychol. Rev.* 69: 74–90, 1962.
35. Widdowson, E. M. and McCance R. A.: Some effects of accelerating growth. General Somatic development. *Proc. Roy. Soc.* 152: 199–206, 1960.

Chapter 24

SUGAR INGESTION AND CARIES

Rachel H. Larson

The ingestion of sweet tasting foods and their relation to the development of dental caries has been a matter of concern since the time of Aristotle.[29] Hardwick [7] reports that by 1600, it had already become apparent to English observers that the use of "most confections and sugar plummes heateth the blood, roteth the teeth and maketh them look black."

In spite of these early references to dental caries, relatively few fossil teeth show cavitation, and it is generally accepted that dental caries is a disease of modern civilized man, which becomes universally present when his diet becomes ample and refined. Several reports [28,32] have shown caries incidence to be extremely low in most primitive populations existing without refined carbohydrates whether their diets consisted mainly of fats and proteins as the Eskimos, or of starchy cereal and vegetables as some African tribes.

CARIES LEVELS AND SUGAR INTAKE

The diet, eating habits and the whole environment of modern man is so different from those of primitive man that the conclusion cannot be drawn that sugar alone is responsible for the high levels of caries currently experienced. However, in our population there are many examples of groups or individuals whose high levels of caries can be directly related to sugar intake. Miller [25] reported unusually severe dental caries in confectioners, pastry bakers and other individuals who presumably had consumed large quantities of sugar. Bibby [1] drew attention to two groups of children who regularly developed low levels of caries: diabetics on low sucrose diets and institutionalized children with limited between-meal snacks of sweet foods. Also of special interest are reports [4,24] of very low levels of caries in fructose-intolerant individuals. This disease is evident from early childhood and is characterized by alarming symptoms of nausea, vomiting, pallor and eventual unconsciousness following the intake of sucrose or other fructose-containing foods. Eeg-Larsen's [4] report indicates that fructose-intolerant individuals do seem to avoid sucrose-containing foods and limit their intake of fruits and vegetables. A decrease in caries was reported following periods of sugar rationing in both Europe [33] and Japan [31] during World War II and increases were experienced when rationing ended. These correlations are suggestive but must not be taken as proof of a causal role of sucrose in caries. The absence or

reduction of sugar in the diet is of necessity accompanied by an increase in some other component which may have a protective effect.

Caries development is dependent upon the interaction of a number of factors, and a high level of sugar consumption is not always associated with a high level of caries development. The Vipeholm study in Sweden [6] reported widely different levels of caries for human subjects consuming high levels of sugar under different conditions. Subjects consuming as much as 300 grams of sugar per day in solution at meals showed no increase in dental caries activity. Whereas, subjects consuming a much lower level of sugar between meals in a sticky form such as toffies and caramels developed caries. When sugar was taken only with the meals and followed by some form of cleaning the teeth, caries development was minimized. This suggests that caries is related not to total ingestion of sugar, but to the amount which remains in the mouth.

CONTROLLING FACTORS IN CARIES DEVELOPMENT

In an attempt to understand some of these data, let us examine the three factors essential for caries development: (a) a supporting dietary substrate, (b) a causative microflora, and (c) susceptible host and teeth. Modification of any one of these three factors may prevent caries development.

Dietary substrate

Fermentable dietary carbohydrates are necessary for acid production and eventual caries development. A number of workers have compared the effect of sucrose with other sugars and with corn starch on caries development in experimental animals. Frostell et al.,[5] Larje and Larson,[15] and Huxley [9] compared levels of caries using experimental conditions under which rats develop both smooth surface and sulcal lesions, reported lower levels of caries in the groups on diets containing glucose than sucrose; differences were more pronounced on the smooth surfaces than in the sulci. In gnotobiotic rats using a model associated with the development of sulcal lesions only, Rosen [27] reported that glucose supported a higher level of caries than sucrose. These findings suggest that organisms which are active in the sulci are less dependent upon sucrose than those active on the smooth surfaces.

Krasse [13,14] reported high levels of implantation and recovery of streptomycin-resistant caries-inducing streptococci and high levels of caries in hamsters fed a sucrose diet. When sucrose was replaced by glucose in the experimental diet very few caries-active organisms were recovered and few carious lesions developed. When cornstarch was substituted for sucrose in a diet normally associated with high levels of caries, even animals exposed to a broad range flora, developed only occasional lesions and these were largely in the sulci (Larson, unpublished). These results are in keeping with Marthaler's [24] studies of patients with hereditary fructose intolerance, and Russell's [28] work with

primitive populations on high starch diets. In each case extremely low levels of caries were observed in spite of poor oral hygiene.

Oral microflora

Microorganisms are essential to caries development. Germfree animals display a complete absence of caries even when maintained on high sucrose diets.[26] Treatment with a broad range antibiotic that depresses caries active flora dramatically reduces the level of caries for animals from a caries active colony fed a high sucrose diet.[23]

Much is known concerning the microorganisms which comprise the oral flora, nevertheless their mechanism of action in caries development is not fully understood. Anything more than a superficial comment is beyond the scope of this presentation. Theoretically, any microorganism which remains adjacent to the tooth surface and produces acid can participate in the development of caries. A wide range of such organisms are apparently active in the molar pits and fissures when they are trapped there along with food particles. However, on the smooth surfaces adhesion is necessary in order to hold acid-producing bacteria in close proximity to the tooth surface. Certain acidogenic organisms of the streptococcal group, including *Streptococcus mutans* are characterized by their ability to produce extracellular polysaccharides. Sucrose has been shown to be specific for extracellular polysaccharide formation *in vitro,*[10] and in rat [5] and hamster.[3,5] However, not all potentially caries-inducing organisms have the same degree of specificity as to carbohydrate requirements. The adhesion of these organisms may be facilitated by these "sticky" polysaccharides, as well as interaction of bacteria with bacteria, enamel or pellicle. These adherent streptococci play a major role in the development of smooth surface lesions and possibly fissure lesions.

The distribution of these organisms in animals and in man is ill defined. The elegant work of Keyes [11] clearly demonstrated that these organisms are not ubiquitous in rodents. He showed that certain colonies of rats and hamsters which were essentially caries-free even on a caries-test diet developed high levels of caries when caged with animals from a caries active colony. Failure to recognize the importance of the microflora in animal studies led to faulty evaluations of the relative caries susceptibility of two colonies of rats. Workers at both Harvard [34] and Michigan Universities [8] by selective breeding established strains of animals which were considered as "caries susceptible" and "caries resistant" because of their response to a given diet and their indigenous flora. In later studies at the National Institute of Dental Research when these animals were infected with the same broad range flora and fed the same diet, both strains from the two colonies developed high levels of caries. Thus, both the Harvard [16] and Hunt-Hoppert [20] strains of rats were mislabelled as "resistant" when the characteristically low levels of caries were actually due more to the lack of caries-inducing organisms than to any genetic resistance *per se.* However, it appears that the absence of caries in man is not due to the absence of potentially caries-inducing organisms. Bratthall [2] has demonstrated the

presence of *S. mutans* in representative populations throughout the world. Furthermore, Stiles et al.[30] have shown that even caries-free populations in certain remote areas with little or no access to sucrose also harbour *S. mutans.*

Host and teeth

Susceptible host and teeth are essential to caries development. Unfortunately most teeth are susceptible to caries development if a sufficient cariogenic challenge is provided. Increased resistance of teeth has been suggested in relation to a number of factors.[35] True differences can be demonstrated only when cariogenic challenges of the same intensity can be provided, which is not possible *in vivo*.[17] However, there is considerable evidence from animal data to suggest that resistance increases with certain morphological characteristics, enamel maturation [18] and increased fluoride uptake.[21]

Host characteristics or habits which modify the cariogenic challenge are much more important than relative susceptibility of the teeth *per se.* Osborne Mendel and NIH Black rats maintained on the same caries test diet and infected with cariogenic organisms developed widely different levels of caries.[19,22] It was then shown that the Black rats which developed low levels of caries ate only a few large "meals" each day, whereas the Osborne Mendel rats which developed high levels of caries nibbled frequently throughout the day. When animals of both strains were placed in automatic feeding machines and forced to eat 36 "meals" per day both strains of animals developed high levels of caries.[12]

SUMMARY

Both human and animal data show that the ingestion of sugar plays a critical role in the development of dental caries. However, the amount consumed is less important than the frequency and duration of oral exposure, the virulence of the oral flora, and the resistance of the teeth which may be influenced by such factors as age, and fluoride uptake.

REFERENCES

1. Bibby, B. G. Inferences from naturally occuring variations in caries prevalence. *J. Dent. Res.* 49:1194–1199, 1970.
2. Bratthall, D. Demonstration of *Streptococcus mutans* strains in some selected areas of the world. *Odont. Rev.* 23:401–410, 1972.
3. Campbell, R. G., and Zinner, D. D.: Effect of certain dietary sugars on hamster caries. *J. Nut.,* 100: 11–20, 1970.
4. Eeg-Larsen, N.: The role of carbohydrates and sugar in dental caries. Presented at the symposium on *The Role of Sugar in Modern Nutrition,* Sundbyberg, Sweden, 1973.
5. Frostell, G., Keyes, P. H. and Larson, R. H.: Effect of various sugars and sugar substitutes on dental caries in hamsters and rats. *J. Nut.,* 93: 65–76, 1967.
6. Gustafsson, B. E., Quensel, C. E., Swenader Lanke, L., Lundqvist, C., Grahnen, H., Bonow, B. E. and Krasse, B.: The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. *Acta Odont. Scand.,* 11: 232–364, 1954.
7. Hardwick, J. L. The incidence and distribution of caries throughout the ages in relation to the Englishman's diet. *Brit. Dent. Jour.* 108: 9–17, 1960.

8. Hunt, H.R., Hoppert, C.A. and Erwin, W.G.: Inheritance of susceptibility to caries in Albino rats (*Mus norvegicus*). *J. Dent. Res.* 23: 385–401, 1944.
9. Huxley, H.G.: The cariogenicity of various percentages of dietary sucrose and glucose in experimental animals. *New Zealand Dental Journal* 67: 85–98, 1971.
10. Jordan, H.V. and Keyes, P.H.: *In vitro* methods for the study of plaque formation and carious lesions. *Archs. Oral Biol.,* 11: 793–802, 1966.
11. Keyes, P.H.: The infectious and transmissible nature of experimental dental caries. Findings and implications. *Archs. Oral Biol.* 1: 304–320, 1960.
12. König, K.G., Larson, R.H. and Guggenheim, B.: A strain-specific eating pattern as a factor limiting the transmissibility of caries activity in rats. *Archs. Oral Biol.* 14:91–103, 1969.
13. Krasse, B.: The effect of the diet on the implantation of caries-inducing streptococci in hamsters. *Archs. Oral Biol.* 10:215–221, 1965.
14. Krasse, B.: The effect of caries-inducing streptococci in hamsters fed diets with sucrose or glucose. *Archs. Oral Biol.* 10:223–226, 1965.
15. Larje, O. and Larson, R.H.: Reduction of dental caries in rats by intermittent feeding with sucrose substitutes. *Archs. Oral Biol.* 15:805–816, 1970.
16. Larson, R.H.: Response of Harvard caries-susceptible and caries-resistant rats to a severe cariogenic challenge. *J. Dent. Res.* 44:1402–1406, 1965.
17. Larson, R.H.: Tooth age and caries susceptibility. In Harris, R.S. (Ed.), *Art and Science of Dental Caries Research,* New York, Academic Press, 1968, pp. 257–275.
18. Larson, R.H. and Fitzgerald, R.J.: Caries development in rats of different ages with controlled flora. *Archs. Oral Biol.* 9:705–712, 1964.
19. Larson, R.H. and Goss, B.J.: Diet as a limiting factor in the transmissibility of caries activity between rats of different strains. *Archs. Oral Biol.* 12:1085–1093, 1967.
20. Larson, R.H., Keyes, P.H. and Goss, B.J.: Development of caries in the Hunt-Hoppert caries-susceptible and caries-resistant rats under different environmental conditions. *J. Dent. Res.* 47:704–709, 1968.
21. Larson, R.H., Mellberg, J.R., Englander, H.R. and Senning, R.S.: Caries inhibition in the rat by water-borne and enamel-bound floride. *Caries Res.* in press.
22. Larson, R.H. and Simms, M.E.: Genetic and Environmental influences on dental caries in the Osborne Mendel and the NIH Black Rat. *Archs. Oral Biol.* 10:663–668, 1965.
23. Larson, R.H. and Zipkin, I.: Effect of tetracycline on the transmission of dental caries in rats. *J. Dent. Res.* 40:264–267, 1961.
24. Marthaler, T.M. Epidemiological and clinical dental findings in relation to intake of carbohydrates. *Caries Res.* 1:222–238, 1967.
25. Miller, W.D. *Micro-Organisms of the Human Mouth.* Philadelphia, S.S. White Dental Manufacturing Co., 1890.
26. Orland, F.J., Blaney, J.R., Harrison, R.W., Reyniers, J.A., Trexler, P.C., Ervin, R.F., Gordon, H.A. and Wagner, M.: Experimental caries in germ-free rats inoculated with enterococci. *J.A.D.A.* 50:259–272, 1955.
27. Rosen, S. Comparison of sucrose and glucose in the causation of dental caries in gnotobiotic rats. *Archs. Oral Biol.* 14:445–450, 1969.
28. Russell, A. L.: Carbohydrates as a causative factor in dental caries epidemiological evidence. In Sipple, H. I. and McNutt, K. W. (Eds.), *Sugars in Nutrition,* New York, Academic Press, 1974, pp. 635–644.
29. Stephan, R. M.: Relative importance of polysaccharides, disaccharides and monosaccharides in the production of caries. *J. A. D. A.,* 37: 530–536, 1948.
30. Stiles, H. M., Donnelly, C. J., Thomson, L. A. and Brunelle, J. A.: A population with limited Western Civilization contact-*Streptococcus mutans* distribution. Preprinted Abstracts, Int. Assoc. Dent. Res. #681, p. 217, March, 1972.

31. Takeuchi, M. Epidemiological study on relation between dental caries incidence and sugar consumption. *The Bulletin of Tokyo Dental College* 1: 58–70, 1960.
32. Toverud, G.: Introductory Review, in *Survey of the Literature of Dental Caries.* Publication 225, National Academy of Sciences, 1952, pp. 3–53.
33. Toverud, G., Rubal, L., and Wiehl, D. G.: IV. The influence of war and postwar conditions on the teeth of the Norwegian school children. *The Millbank Memorial Fund Quarterly,* 39: 489–539, 1961.
34. Willett, N. P., Resnick, J. B. and Shaw, J. H.: A comparison of the salivary protease activity in the Harvard and Hunt-Hoppert Caries-resistant and Caries-susceptible rats. *J. Dent. Res.* 37: 930–937, 1958.
35. Wolstenholme, G. E. W. and O'Connor, M. (Eds.): *Caries-Resistant Teeth,* Boston, Little Brown and Co., 1965.

DISCUSSION

STELLAR: What is the hope of eliminating the microflora? That is the easiest way out for most people.

LARSON: Elimination of the oral flora is probably neither possible nor desirable. Good oral hygiene can reduce the incidence of caries by making changes in the flora. In some cases of rampant caries antibiotics have been used effectively. I can envision the time when some antibiotics, not reserved for more serious diseases, may be widely used against caries.

PFAFFMANN: I have a question about fructose intolerance. You say that such individuals do not have caries. Is that because they do not take sugar?

LARSON: The intolerance is specific for the fructose molecule, but since the disaccharide sucrose releases a molecule of fructose during digestion, fructose intolerant individuals become ill if they consume either fructose or sucrose. One report [25] indicates that not only do these individuals have diets low in prepared foods containing sucrose, but also that they consume rather small amounts of fruits and vegetables. Since their response to fructose leads them to eliminate essentially all sugars from their diet they typically have a low level of caries. Dr. Muhlemann would you care to comment?

MUHLEMANN: They eat a lot of bread, spaghetti, noodles, everything but no sucrose, no fructose. Their brothers and sisters who do not have the intolerance have a lot of dental caries.

STELLAR: Is that an enzymatic matter?

MUHLEMANN: Yes. Liver aldolase deficiency.

LARSON: There is one report of a physician who did not recognize for many years that his son was fructose intolerant. He thought the boy was allergic and that he would outgrow it. He attempted to get him over this supposed allergy by forcing him to eat some sucrose after every meal. He would have him eat as much as he could tolerate. The boy continued to get sick and, in addition, developed caries because of the sugar. Perhaps he should have sued his father for malpractice.

JACOBS: As in the case of cigarettes and cancer, progress in hypothesizing causal mechanisms resulted when

simple "per capita consumption" data began to be broken down by age, heavy vs. light smokers, and other individual parameters. In the case of sucrose ingestion, market data on "per capita consumption" over the years have suggested an association between caries and sucrose "consumption." Your reference to the Vipeholm study in Sweden,[6] and your own work on rats,[12,19,22] suggests that increased "snacking" with sucrose, *without opportunity to rinse the mouth,* is a critical factor in caries, i.e., that it is more complicated than correlating total "per capita consumption" with incidence of caries.

Would you agree that the role of food habits in caries will be increasingly understood by continuing to fill the gap between the animal work, where individual intake can be measured, and "per capita consumption" based upon economic indicators of productivity and population estimates from census data?

LARSON: Your point is well taken. Clearly, quite different kinds of information come from studies in which countries are the unit of analysis as in the "per capita" studies and cases in which groups of similar individuals experiencing different conditions are compared. The Vipeholm study demonstrates what can be done to explore mechanisms of caries production in humans directly. Perhaps we can fill the "gap" to which you refer from both sides.

PART VI
COMMENTS AND PERSPECTIVES

Chapter 25

SWEET PREFERENCE AND THE SENSORY CONTROL OF CALORIC INTAKE

Jacques Le Magnen

In reviewing the literature on "Sweet preference" for the preparation of this paper, the author was again impressed by three facts: First, this topic which may appear very limited and therefore simple to a naive person is of an extraordinary complexity. Second, the accumulation, during the last 25 years, of much beautiful data including the replication of the same, or almost the same, experiments by many investigators, has increased, rather than decreased the difficulties in reaching a general consensus. Third, experimental data are useful in understanding and solving a problem only if they are included in a body of clear and unequivocal concepts. Words, such as taste preference and aversion, pleasantness and unpleasantness, hedonic value, palatability and reactivity to taste, represent overlapping and obviously related concepts. The concept of taste preference is particularly ambiguous and confusing: preference to what? and in doing what?

MEASUREMENT

As is generally the case in physiological studies of behavioral responses, the main source of ambiguity is the difference between the measurement of phenomena in human and animal subjects. Psychophysicists have shown that humans can quantify the pleasantness evoked by a sensory stimulus separately from its quality and intensity. Furthermore, such judgements can be made in the absence of any observable concurrently elicited behavior. The human hedonic response is thus a subjective and absolute judgement. It is, more or less, postulated by psychophysicists, that the response is a reliable measure of one of the dimensions of a sensation. Thus hedonic judgements, like judgements of intensity or quality, would reflect the response of the sensory apparatus to intrinsic properties of the stimulus.

This view of judgements has, for a long time, contaminated the conceptual treatment of presumed homologous phenomena experimentally approached by very different measures, in rats. When the rat is questioned by the presentation of a sweet solution, the answer is generally the amount drunk in a given time, compared to amounts drunk or eaten of other items simultaneously or identically offered. However, this measure of "preference," the drinking re-

sponse, is relative to the modality of presentation and to the alternatives offered. Nonetheless, sensory physiologists have often been tempted to postulate that the ingestive response is entirely determined by sensory activity and therefore, like the human judgement, reliably measures an intrinsic property of the stimulus. In fact, the problem is really one of knowing the extent to which the palatability of a particular solution determines the drinking response to that fluid under various conditions. The question of whether sweet solutions are palatable to rats becomes the question of when and how palatability *per se* affects intake?

PREFERENCE FOR SWEETNESS

Instead of pointing out the discrepancies (particularly of interpretation) which remain in the literature, I will try to sum up the experimental data and suggested mechanisms on which a general agreement seems to have been reached.

The stimulus

Everybody has noted the various properties of a sweet solution of glucose or sucrose. It is a liquid that is the source of some specific mechanical stimuli in the mouth and as such elicits a specific consummatory response named a drinking response. It is sweet, this sweetness being dependent both on the particular sugar and on its concentration. It is a source of calories. It is a liquid with a definite osmotic pressure. Finally, it is an aqueous solution and thus a source of water. The situation is not simple. Fortunately, it is possible to experimentally contrast sweet fluids with other fluids having in common some of these properties, and not others, essentially with saccharine solution, salty solutions and water.

Sweet preference, the enhancement of a defined drinking response by the sweet taste of a solution, is both sensory specific and hunger dependent. To assert that higher responding is due to sweet preference requires several types of supporting evidence. The responding must be shown to be dependent on the sweetness of the solution. Thus, it is not exhibited under the same conditions for fluids of other tastes (salt solutions and water), and has some specific relation to the intensity of the stimulus. In addition, the response must be shown to be dependent on the solution's sweetness rather than its caloric value, its osmolarity or its water content. Finally, the sweet specific response must be shown to vary with hunger, that is previous food deprivation. Food deprivation is assumed to influence the response to sweet nutritive or non-nutritive solutions but not the response to saline or water. Conversely, water deprivation is assumed not to influence the intake of sweet solutions but to have a specific effect on the intake of water and salt solutions.

The role of sweetness

Much evidence has been provided that rats offered a fluid under specified conditions for a given time enhance their intake of that fluid, only if it is, and because it is,

sweet. The works of Young and coworkers,[36,37,38] of Hagstrom and Pfaffmann,[14] Jacobs,[16,17,18] Collier [7] and of many others, have provided convincing evidence that ingestion of sweet solutions varies as a function of concentration and therefore of stimulus intensity. It also has been demonstrated that isosweet solutions of different sugars yield identical rates of short term intake [29] and are equally reinforcing during the acquisition of an instrumental response.[12,13] It is significant that saccharine solutions, like sugar solutions, are palatable and that their palatability is related to concentration. Furthermore, saccharine on the basis of its sweet taste alone is reinforcing in an instrumental learning situation.[28]

The role of hunger

The dependence of sweet preference on hunger is reflected in the increased palatability of sweet solutions as a function of food deprivation and has been recognized for a long time.[36,37,38,7,4,25,5,30,31,32] That such increments in response are independent of caloric content is clearly demonstrated by the, so called, "saccharine deprivation effect." [1,33,10] The same food deprivation that increases response to sweet solutions does not affect the response to water, or to solutions having another taste.[36,37,38,9,33,7] But this demonstration is obscured by the well known difficulty in separating food and water deprivation. This difficulty is attenuated by using insulin administration which is generally acknowledged to mimic the humoral conditions of natural hunger (Fig. 25–1). Insulin (3 I.U./Kg body weight) was administered 30 minutes prior to a 30 minute presentation of either a 5% sucrose solution, a 0.037% saccharine solution, a 1% NaCl solution or water. Insulin injected rats, compared to non-injected controls, showed increases in consumption of the two sweet tasting solutions but no changes in the intake of water and saline.[20]

The fact that sweet solutions contain water adds to the difficulty of separating thirst, which should act specifically on water and salt intake, from hunger which modulates preference for sweet. Differential drinking of sweet and non-sweet solutions following manipulation of water imbalance by gastric loading clarifies the issues. Rats that had been pre-loaded with water drank less water, in 30 minute tests, than rats that had received a preload of hypertonic saline (18% NaCl). Animals in both groups drank very small volumes of a hypertonic saline solution but larger and approximately equal volumes of a hypertonic (22%) sucrose and a hypotonic saccharine solution [19] (see Fig. 25–2).

SATIATION

The sensory specificity and hunger dependence of the palatability of sweet solutions are purely exhibited and expressed by the drinking response only in a brief exposure to the solutions. In presentations exceeding 5 to 10 minutes, drinking is increasingly determined by factors other than the initial stimulating effect of the sweet tasting solution. These factors are those involved in the development of satiation during the

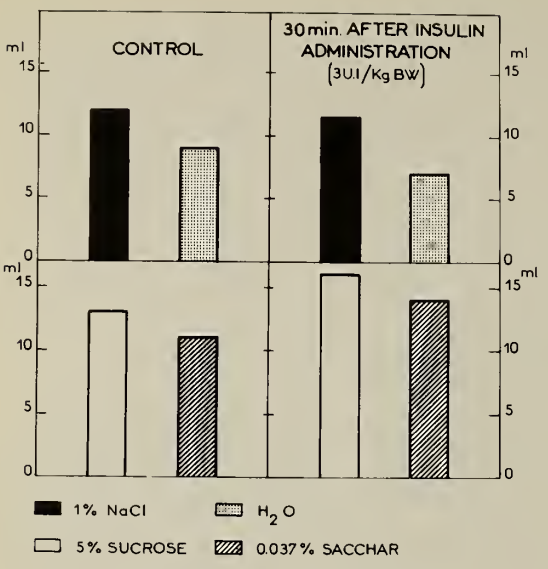


Figure 25–1. Comparison of the effect of insulin-induced hunger on the intake of sweet solutions. salt solutions and water.

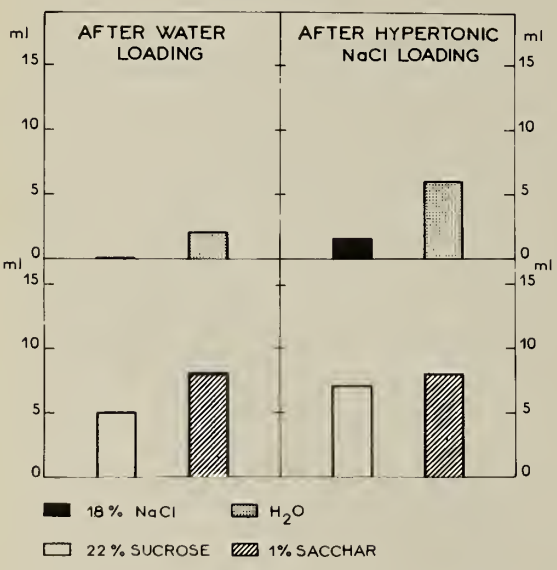


Figure 25–2. Comparison of the effect of water and hypertonic preload on the intake of sweet solutions, hypertonic salt solution or water.

meal. With longer presentations, the cumulative intake becomes increasingly dependent on factors involved in the alteration and time patterning of hunger and satiety that characterize the response to *ad libitum* conditions.

What causes satiation and satiety? An apparently stupid answer is that satiation and satiety are caused by having eaten or drunk. It is is impossible to fully discuss this considerable problem here. Briefly, three sets of ingestive and post-ingestive effects of a food or a fluid are successively active and differently combined over time to suppress hunger and thus to determine the cumulative intake.

1/ The sensory activity of foods and fluids passing via the mouth may itself have a satiating effect. Such a sensory specific oral satiety affects preferences and aversions. In an early demonstration, oral satiety was "conditioned" in rats fed artificially flavored solid diet. After the free intake of the odorized food was repeatedly associated with post-prandial administration of either glucose, insulin or d-amphetamine, reductions and enhancements of food intake appropriate to a "conditioned odor satiety" were observed.[22,23,24] The demonstration of a similar experimentally induced "oral satiety" has recently been extended by Booth and coworkers.[2] Various evidence has been provided that satiation by a sweet solution may include an oral and sensory specific satiety.[27,15]

2/ The contribution of solutions reaching the stomach to satiation has been extensively studied and discussed. Whether gastric chemoreceptors responsive to sweetness are involved or not is uncertain but this possibility has not been excluded.

Although the acknowledged negative feedback from the gastrointestinal tract acting through the vagus nerve on the central nervous system

inhibits hunger, it is clear that current intake remains orally mediated. In other words, the gastric inhibition of the oral response during the meal operates by means of a progressive shift in palatability from the beginning to the end of the free intake.

3/ The most evident effect of post-ingestive factors on cumulative oral intake is due to systemic repletion. It is obvious, and has been clearly demonstrated, that caloric and overall dietary balance plays a part in determining the termination of each meal and in setting the patterns of intake under 24h *ad libitum* conditions.[8,31,16,17,18] The importance of a solution's caloric content and its effect on caloric balance in the 24h intake of a sucrose solution has been clearly shown by Collier.[6] Some problems remain in explaining the 24h *ad libitum* intake of a saccharine solution.[34,35]

LEARNING

The high palatability of sweet solutions for naive subjects and neonates supports the proposition that the sweet taste is an unconditioned stimulus for eating and drinking in the hungry animal. There is other evidence that this behavior is genetically determined. However, it has been clearly shown in the past 15 years that sweet preference, as other innate responses to sensory stimuli, is modified by experience. The conditioned taste aversion by which a sucrose or saccharine solution becomes aversive after a single association with illness or poisoning has been extensively studied.[26,11] This experimental paradigm makes evident a critical physiological process. Taste and smell, food and fluid preferences and aversions, in other words palatability responses, are modulated (as mentioned above) by learning. In this learning the post-ingestive effect of the caloric and dietary balance acts as a positive or negative reinforcer to modulate, to increase or decrease, the palatability of each food item. The first demonstration of this process in the development or suppression of the sweet saccharine preference has been given by Capretta.[5] When daily one hour presentations of saccharine were paired with a gastric load of glucose, the already high intake of a saccharine solution by rats was increased. Pairing saccharine with a water load, decreased intake. The extinction (more exactly, in Pavlovian terms, the "habituation") of the response to saccharine has also been shown in another experiment.[20] For five days, fasting rats received their only calories and fluid, from a daily 3 hour presentation either of sucrose or saccharine solution (Fig. 25–3).[21] The group on sucrose increased their intake of the solution. The group on saccharine at first maintained, and later decreased their consumption of the sweet solution. This extinction of the saccharine preference may be obscured by the reinforcing effect of the Na-saccharine solution on a mixed food and water deprived rat.[32]

The most clear evidence of the physiological conditioning of sweet preference has been recently presented by David Booth.[2,3] He showed that preference for a solution of a particular sweetness was increased by its previous association with post-

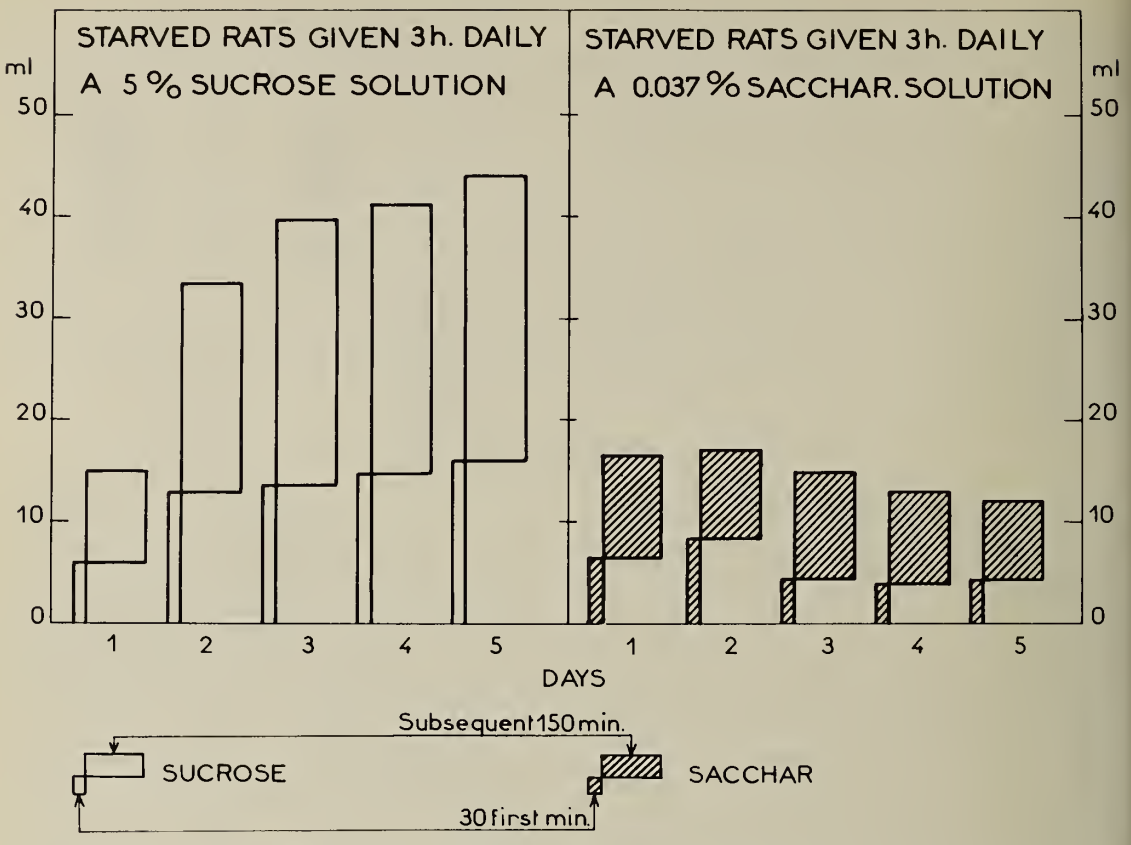


Figure 25–3. Responses of starved rats during 5 consecutive days, to sucrose solution compared to saccharine solution.

ingestive effects of 10% glucose or polysaccharide solutions. Sweetness acceptance was decreased by previous association with 25%-50% glucose solutions. Conditioning, in these two directions simultaneously, by continuous access to a choice of solutions, can reverse the usual gradient of preference for the sweetest solution. The reversal extinguished over a few days without reinforcement.

In summary, a body of convergent experimental results provides conclusive evidence that a complex interaction of three control systems determines the degree to which rats exhibit a sweet preference under various conditions. Initial and unconditioned palatability is modified by post-ingestive effects of repression or facilitation. Some post-ingestive effects are immediate and others are latent. The sweet preference is not a constant property of the stimulus but is essentially relative and undergoes continous adjustment to anticipate the requirements of a behavioral regulation of the nutritive balance.

REFERENCES

1. Bacon, W. E., Snyder, H. L. and Hulse, S. H.: Saccharine preference in satiated and deprived rats. *J. Comp. Physiol. Psychol.*, 55: 112–114, 1952.
2. Booth, D. A., Lovett, D. and McSherry, G.M.: Postingestive modulation of the sweetness preference gradient in the rat. *J. Comp. Physiol. Psychol.*, 78 (3): 485–512, 1972.
3. Booth, D.A. Conditioned satiety in the rat. *J. Comp. Physiol. Psychol.*, 81 (3): 457–471, 1972.
4. Campbell, B.E.: Absolute and relative sucrose preference threshold for hungry and satiated rats. *J. Comp. Physiol. Psychol.*, 51: 795–800, 1958.
5. Capretta, P. J.: Saccharine consumption under various conditions of hunger drive. *J. Comp. Physiol. Psychol.*, 55: 656–660, 1962.
6. Collier, G. and Bolles, R.: Some determinants of intake of sucrose solutions. *J. Comp. Physiol. Psychol.*, 65 (3): 379–381, 1968.
7. Collier, G. and Bolles, R.: Hunger and thirst and their interaction as determinants of sucrose consumption. *J. Comp. Psychol.*, 66(3): 633–641, 1968.
8. Davis, J. D.: The effectiveness of some sugars in stimulating licking behaviour in the rat. *Physiol. and Behav.*, 11 (1): 39–45, 1973.
9. Ernits, T. and Corbit, J. D.: Taste as a dipsogenic stimulus. *J. Comp. Physiol. Psychol.*, 83 (1): 27–31, 1973.
10. Fuller, J. L.: Genetic aspects of regulation of food intake. *Advances in Psychomatic Medicine:* Hunger and satiety in health and disease, vol. 7., ed. Reischman, 1972, pp. 2–24.
11. Garcia, J. and Ervin, F. R.: Gustatory-visceral and tele-receptor cutaneous conditioning adatation in internal and external milieus. *Communic. in Behav. Biol.*, 1: 389–415, 1968.
12. Gutman, N.: Operant conditioning extinction and periodic reinforcement in relation to concentration of sucrose used as reinforcement agent. *J. Exper. Psychol.*, 46 (4): 213–214, 1953.
13. Gutman, N.: Equal reinforcement value for sucrose and glucose solutions compared with equal sweetness values. *J. Comp. Physiol. Psychol.*, 47: 348–361, 1954.
14. Hagstrom, E. C. and Pfaffmann, C.: The relative taste effectiveness of different sugars in the rat. *J. Comp. Physiol. Psychol.*, 52: 259–261, 1959.
15. Hsiao, S. and Tuntland, P.: Short-term satiety signals generated by saccharine and glucose solutions. *Physiol. and Behav.*, 7 (2): 287–289, 1971.
16. Jacobs, H. L.: Study on sugar preference: preference for glucose solutions. *J. Comp. Physiol. Psychol.*, 51: 304–310, 1958.
17. Jacobs, H. L.: Some physical metabolic and sensory components in the appetite for glucose. *Amer. J. Physiol.*, 203: 1043–1054, 1962.
18. Jacobs, H. L.: Evaluation of the osmotic effect of the glucose loads on food satiation. *J. Comp. Physiol. Psychol.*, 57: 300–310, 1964.
19. Le Magnen, J.: Nouvelles données sur le processus de régulation des consommations hydrique et saline chez le rat blanc. *C. R. Soc. Biol. Paris,* 147: 1675–1677, 1953.
20. Le Magnen, J.: Activité de l'insuline sur la consommation spontanée de solutions sapides. *C.R. Soc. Biol. Paris,* 147:1753–1757, 1953.
21. Le Magnen, J.: Le processus de discrimination par le rat blanc des stimuli sucrés alimentaires et non alimentaires. *J. Physiol. Paris,* 46: 414–418, 1954.
22. Le Magnen, J.: Sur le mécanisme d'établissement des appétits caloriques. *C. R. Acad. Sci. Paris,* 240: 2436–2438, 1955.
23. Le Magnen, J.: Efficacité des divers stimuli alimentaires dans l'établissement et la commande d'un appétit chez le rat blanc. *J. Physiol. Paris,* 51: 987–998, 1959.
24. Le Magnen, J.: Perispheral and systemic actions of food in the caloric regulation of intake. *Ann.N.Y.Acad.Sci.*, 157:1126—1157, 1969.

25. Mook, D.G.: Some determinants of preference and aversion in the rat. *Ann. N. Y. Acad. Sci.,* 157:1158–1175, 1969.
26. Nachman, M.: Learned taste and temperature aversions due to lithium chloride sickness after temporal delays. *J. Comp. Physiol. Psychol.,* 73: 22–30, 1970.
27. Schulz, T.W. and Lawrence, D.H.: Learning of a discrimination in satiated and deprived rats with sucrose as incentive. *Amer. J. Psychol.,* 71: 563–567, 1958.
28. Shefield, F.D. and Roby, T.B.: Reward value of a non-nutritive sweet taste. *J. Comp. Physiol. Psychol.,* 43:471–481, 1950.
29. Shuford, E.H.: Palatability and osmotic pressure of glucose and sucrose solutions as determinants of intake. *J. Comp. Physiol. Psychol.,* 52:150–153, 1959.
30. Smith, M.P. and Capretta, P.J.: Effects of drive level and experience on the reward value of saccharine solutions. *J. Comp. Physiol. Psychol.,* 49: 553–557, 1956.
31. Smith, M.P. and Duffy, M.: Consumption of sucrose and saccharine by hungry and satiated rats. *J. Comp. Physiol. Psychol.,* 50:65–69, 1957.
32. Smith, M.P. and Duffy, M.: Some physiological factors that regulate eating behavior. *J. Comp. Physiol. Psychol.,* 50:601–608, 1957.
33. Strouthers, A.: Thirst and saccharine preference in rat. *Physiol. and Behav.,* 6 (4): 289–292, 1971.
34. Wagner, M.W.: Satiation effects on rate of intake and preference of glucose solutions. *J. Comp. Physiol. Psychol.,* 59 (1): 115–117, 1965.
35. Warren, R.P. and Warren, R.M.: Soluble saccharine preference: a nutritive basis for persistence. *Nature,* 210 (5033): 310–311, 1966.
36. Young, P.T. and Greene, J.T.: Quantity of food ingested as a measure of relative acceptability. *J. Comp. Physiol. Psychol.,* 46:288–294, 1953.
37. Young, P.T. and Greene, J.T.: Relative acceptability of saccharin solutions as revealed by different methods. *J. Comp. Physiol. Psychol.,* 46: 295–298, 1953.
38. Young, P.T. and Falk, J.L.: Relative acceptability of NaCl solutions as a function of concentration and water need. *J. Comp. Physiol. Psychol.,* 49: 569–575, 1956.

Chapter 26

SWEET PREFERENCE AND HEDONIC EXPERIENCE

Eliot Stellar

As many of the papers in this conference show, we infer *sweet preference* from a variety of different measurements that we make in both animals and man. We infer it from the high levels of ingestion of sugars and other sweet substances. We infer it from choices organisms make in favor of sweet substances, sometimes in situations where they can ingest a great deal, sometimes where they can only "taste" and ingest very little. We also infer it in situations where the organism works for the "sweet taste" that it gets, performing many instrumental responses (e.g., lever presses) to obtain very small quantities of "taste." In this case, when he works harder, we say he is more strongly motivated or has a stronger sweet preference. Finally, we measure sweet preference in man by asking him to rate the hedonic quality of a taste substance on a rating scale or to describe the pleasantness or unpleasantness he experiences upon tasting.

While all of these measures reveal the existence of sweet preference, it is no surprise that different methods of measurement yield different results. Often this is because the different methods of measurement reveal the operation of different parts of the very complex mechanism that produces sweet preference. In the experiments, from our laboratory, that I will report here, we have tried to separate oral from gastric and other postingestional factors. We have tried to identify the operation of oral factors other than taste, we have tried to take into account the state of the animal (e.g. hunger, thirst) at the time of measurement, and we have recognized the importance of learning and habit in the genesis and maintenance of sweet preference.

The animal and human subjects of our experiments are all adults, but I believe that insofar as our findings give us insight into the nature of preference they can contribute to the understanding of the genesis of sweet preference in the developing organism. Let me turn to a review of our data.

PREFERENCE-AVERSION IN NORMAL RATS

The first Figure shows the reference-aversion curves in the rat for glucose and fructose that McCleary [7] obtained by the single stimulus method. The rats are 16 hours thirsty and each day given one hour to drink water or some concentration of glucose or fructose.

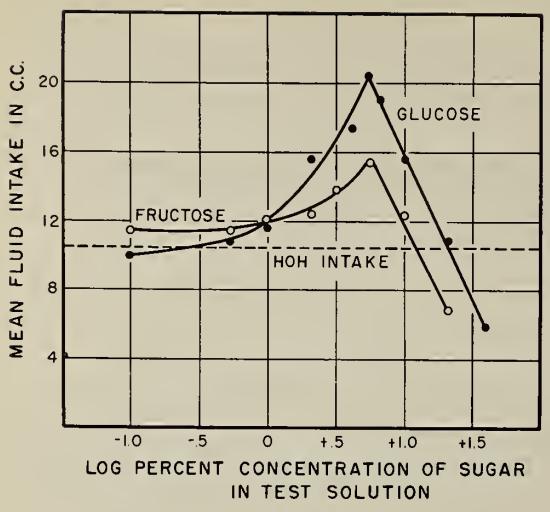


Figure 26–1. Preference-aversion curves in the rat for glucose and fructose by the single-stimulus, one-hour ingestion method.

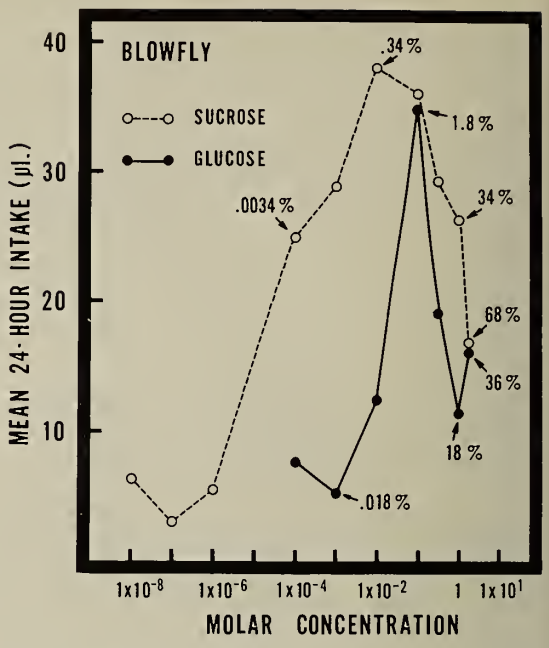


Figure 26–2. Preference-aversion curves in the blowfly for sucrose and glucose by the 2-choice, 24-hour ingestion method.

Amount ingested is taken as the measure of preference. As you can see, the rising and falling preference-aversion curves have the same shape as those obtained when the rats are given a choice and have 24-hour access to 2 bottles, one containing water and the other some concentration of a sugar as in the experiments of Richter.[10] However, peak preference is at 5% concentration in the single stimulus method and at 10% in the 2-bottle choice method. The reason is, as you will see, that the dehydrated rat in the one-hour, single-stimulus test cannot drink as much hypertonic solution as the rat in water balance (24-hour, 2-bottle test).

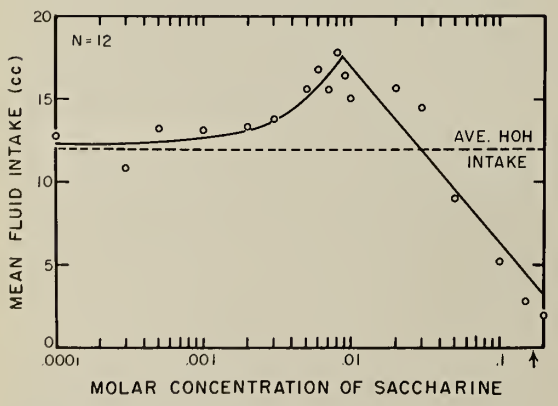


Figure 26–3. Preference-aversion curve in the rat for saccharine by the single-stimulus, one-hour ingestion method. Arrow on absicca shows isotonic concentration.

An even more important fact, common to both methods, is that the animals drink so little of the hypertonic solutions and, indeed, are averse to them in comparison to water. The aversion turns out to be due, in large part, to postingestional factors, working together with oral factors. This interaction of oral and postingestional factors is beautifully illustrated in the experiments of Dethier[2] on the blowfly. In this organism, the intensity of drinking is a direct function of the intensity of sugar concentration. When the foregut becomes loaded,

however, then drinking is inhibited, turned off. The resultant preference-aversion function for glucose and sucrose are illustrated in Figure 26–2. If the recurrent nerve that signals when the foregut is loaded is severed, then there is no inhibition and the fly keeps drinking, driven by the taste stimulus, and literally may burst.

That the rising and falling preference-aversion curve may be due entirely to positive and negative taste factors is suggested by experiments that we did with saccharine.[12] The whole preference-aversion function is completed before the fluids reach isotonic concentration (see arrow on abscissa of Figure 26–3). Thus, in this case, postingestional osmotic factors are not likely to play a role. On the other hand, there is direct evidence that aversion to strong sugar solutions is due to a postingestional factor. When McCleary [7] preloaded rats with hypertonic solutions of either sugar or NaCl before the one-hour ingestion test, they decreased their drinking of hypertonic solutions and increased their water drinking (Fig. 26–4). The changes in drinking are not specifically dependent on gastric effects of the preload since similar results are obtained in the rabbit when hypertonic NaCl preloads are given intravenously (Fig. 26–5). In fact, it turns out that the rats' drinking of hypertonic sugar solutions can be decreased by simply increasing their thirst which, of course, also increases their water drinking.[6]

PREFERENCE-AVERSION IN RATS WITH ESOPHAGEAL FISTULAS

An even clearer separation of oral and postingestional factors was obtained by Mook [8] who used the esophageal fistula preparation in single-stimulus testing (Fig. 26–6). In this preparation, the rat's esophagus is cut and brought out at the neck so that nothing it drinks reaches the stomach. A plastic tube can be threaded into the stomach through the lower esophagus so that fluids can be delivered to the alimentary tract.

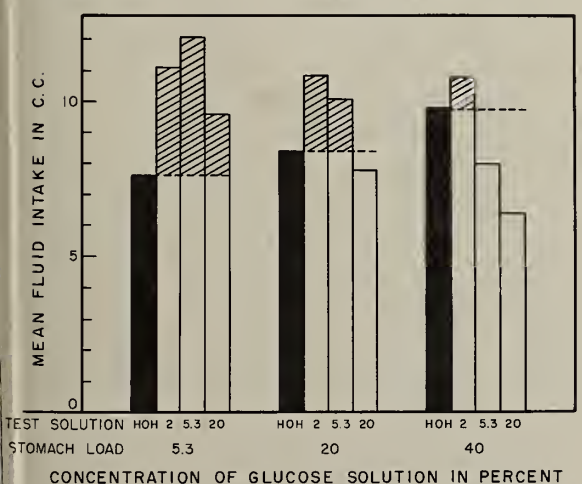

MEAN FLUID INTAKE IN C.C.
10
5
0
TEST SOLUTION HOH 2 5.3 20 HOH 2 5.3 20 HOH 2 5.3 20
STOMACH LOAD 5.3 20 40
CONCENTRATION OF GLUCOSE SOLUTION IN PERCENT

Figure 26–4. Effect of isotonic (5.3%) and hypertonic (20% and 40%) preloads of 3 ml on the ingestion of water, 2%, 5.3%, and 20% glucose by the rat in the one-hour test.

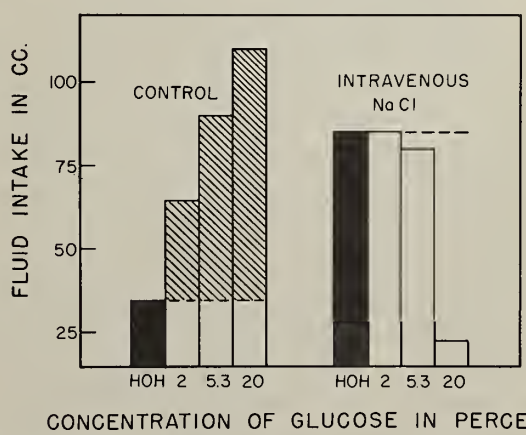


Figure 26–5. Effect of hypertonic (3%) NaCl given intravenously as a preload on the rabbit's drinking of water and glucose solutions.

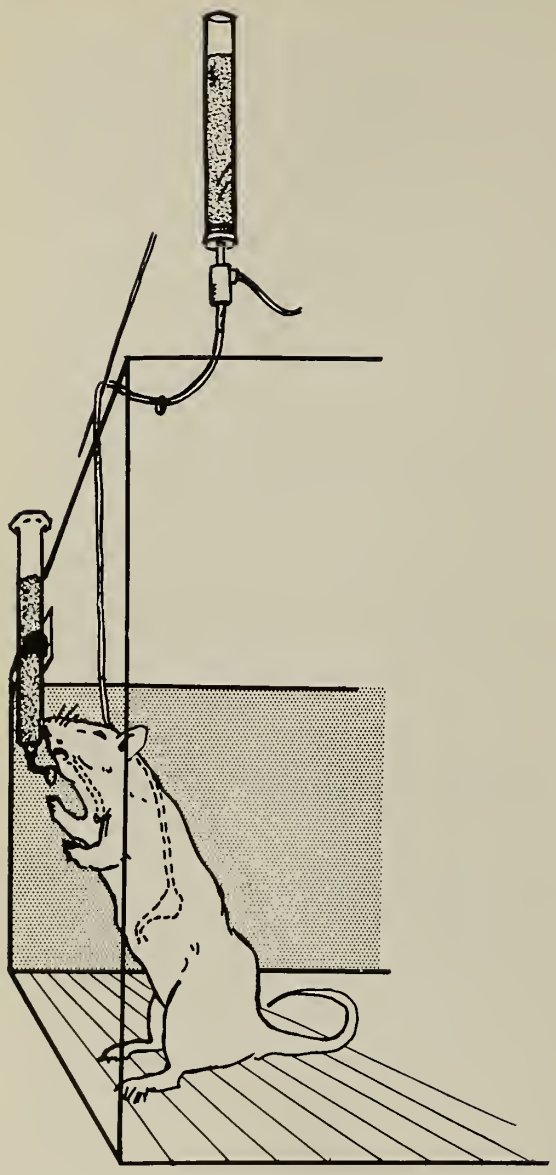


Figure 26–6. Esophageal fistual preparation in which the same or different solutions can be delivered to the mouth and the stomach independently, but simultaneously.

This process can be made automatic by having every 20 tongue laps the rat makes while drinking activate a solenoid valve so as to deliver a measure of fluid to the stomach. This arrangement permits the comparison of sham drinking when nothing reaches the stomach, with normal drinking,

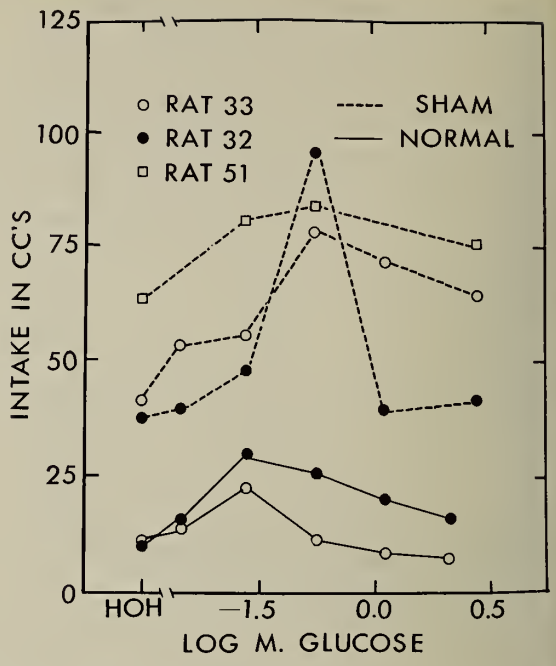


Figure 26–7. Preference-aversion curves for glucose during sham drinking (nothing reaches the stomach) and normal drinking in the esophageal fistual preparation.

when the same fluid that passes through the mouth is simultaneously and synchronously delivered to the stomach. Perhaps this preparation provides the most powerful analysis of all when the rat drinks one substance and gets a different one delivered to its stomach.

As Figure 26–7 shows, the preference-aversion function survives in sham drinking. This suggests a negative oral factor to account for the falling, aversion part of the curve, but as you will see in a moment, it is more likely a negative osmotic, oral factor than a taste aversion or a negative taste factor.

That the postingestional factors operative in the normal preference-aversion function are mainly osmotic is seen in Figure 26–8. Here the animal is drinking the various concentra-

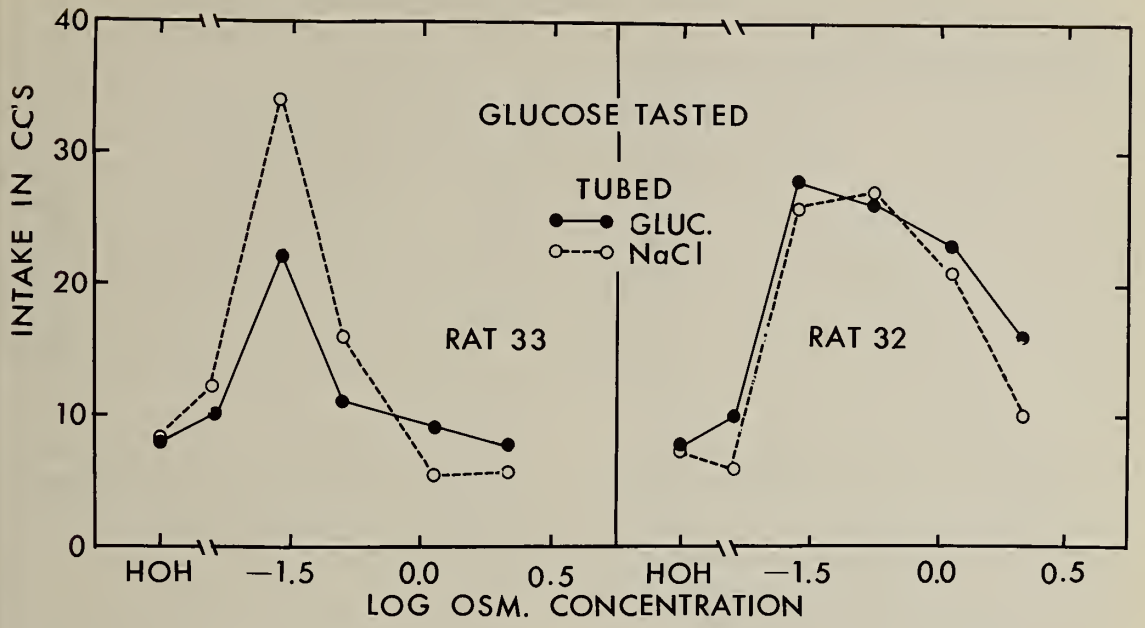


Figure 26–8. Preference-aversion curves for glucose in two esophageal fistual preparations where glucose is tasted and either the same glucose concentrations or osmotically-matched NaCl solutions reach the stomach.

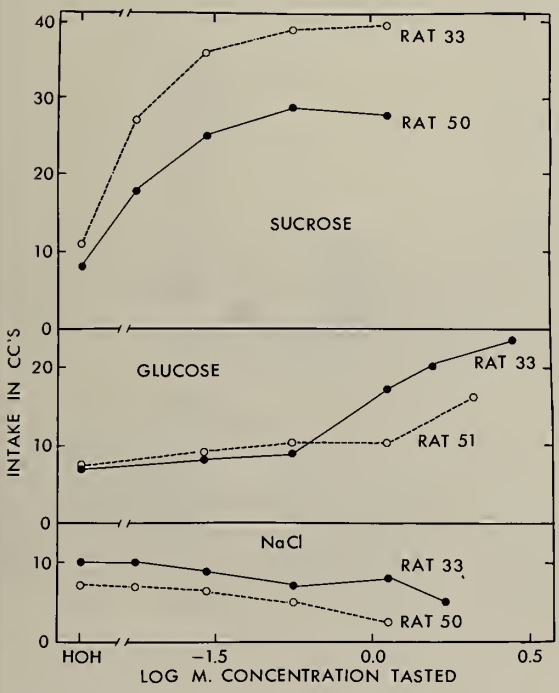


Figure 26–9. Preference-aversion curves in esophageal fistula preparations for sucrose, glucose, and NaCl where only water reaches the stomach.

tions of glucose and is receiving into the stomach either the same concentration of glucose or an osmotically-matched concentration of NaCl. It doesn't matter, the preference-aversion functions are identical in the two cases.

Even more instructive is the case where the animal drinks various concentrations of glucose and sucrose but receives only equal volumes of water in the stomach (Fig. 26–9). The preference-aversion functions are greatly altered. The low concentrations of glucose are no longer preferred. It is not until the hypertonic concentration, normally aversive, that preference is shown. Sucrose, on the other hand, shows a strong preference function at the lower concentrations, but no aversion or falling function at the high concentrations.

If you remember that the rat is 16-hours thirsty and in the single-stimulus testing situation, it is possible to make sense out of these findings. The preference for the low concentrations of glucose is not necessarily a taste preference, but may be a "dilute water effect." That is to say, thirsty rats, forced to drink glucose solutions, must drink greater volumes of more concentrated solutions to get the same amount of water in excess of solute. When this situation is corrected by putting water in the stomach as the rat drinks glucose, there is nothing left to motivate excess drinking. On the contrary, with sucrose, the preference remains at low concentrations, suggesting that it is a true taste preference. This idea is strongly supported by the data of Hagstrom and Pfaffmann [3] who recorded from the taste nerves of the rat and found that at the hypotonic concentrations glucose was hardly stimulating at all while sucrose was very stimulating. In fact as Figure 26–10 shows, sucrose yielded the same kind of negatively accelerated function as we obtained in our tests and glucose yielded the same positively accelerated function!

As you can see, any possible negative postingestional effects of the hypertonic concentrations in this particular study were removed by arranging that only water reached the stomach. It is possible that the hypertonic solutions had the same type of negative oral osmotic effects as occur in sham drinking, but these were overridden by an osmotically positive signal from the gut elicited by water. It is as though the dehydrated rat is getting both an early warning, negative osmotic signal from the mouth to "inhibit drinking because this is a hypertonic solution that will further dehydrate you" and a second countermanding signal arising from water in the gut saying: "no, it won't dehydrate you because water is coming down here." If you will pardon the dramatization, it is clear that we have a complex interaction of oral taste and oral osmotic factors with postingestional factors in the rat's preference for sweet.

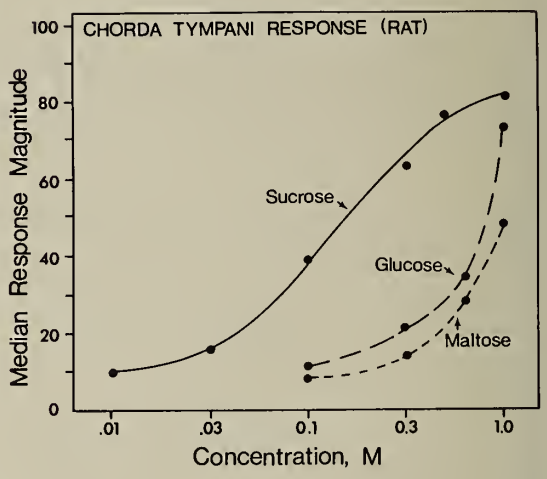


Figure 26–10. Hagstrom and Pfaffmann's data, showing electrophysiological response of taste nerves (chorda tympani) of the rat to different concentrations of sucrose and glucose.

PREFERENCE-AVERSION IN HUMAN SUBJECTS

The same kind of preference for sweet can be studied in the same way by measuring the fluid intake of human subjects, with the added advantage of getting hedonic ratings and reports of preference or pleasantness and unpleasantness, discomfort or nausea. Using the single-stimulus method, Jordan and I [5,13] determined preference-aversion functions in human subjects for glucose, sucrose, saccharine, and sucaryl. We asked subjects not to drink water for several hours, and under this mild dehydration, had them drink fluids from a hidden reservior through a straw for a 10-minute test-period (Fig. 26–11). As Figure 26–12 shows, a

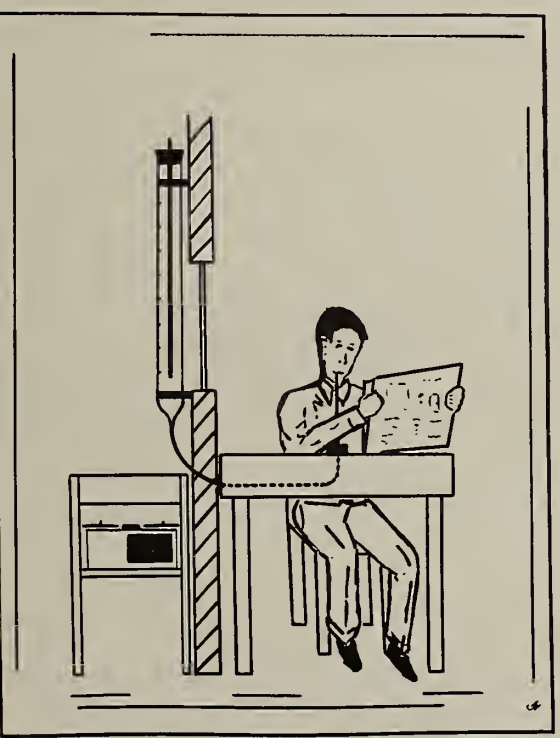


Figure 26–11. Arrangement to test the ingestion of fluids in the laboratory by human subjects where subjects do not see what they are ingesting.

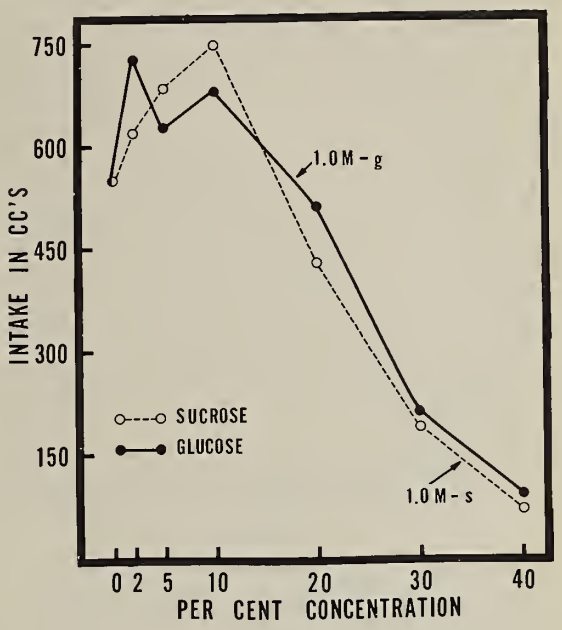


Figure 26–12. Preference-aversion curves in man for sucrose and glucose by the single-stimulus, 10-minute ingestion method.

preference-aversion curve for glucose and sucrose can be generated this way, with subjects showing very strong aversion to the hypertonic concentration and calling them "too sweet" and "nauseatingly sweet" (implying a postingestional effect).

The preference-aversion function for sucrose shows up even more clearly when cumulative intake over ten minutes is shown for the different concentrations (Fig. 26–13). The curve for saccharine is most interesting because it has only an aversion limb in man (Fig. 26–14). The subjects report that as soon as they can taste the sweet, they can taste the bitter and reject it. With sucaryl the story is different (Fig. 26–15), for there is no bitter reported. The aversion to high concentration is explained by the subjects in the same way they explain

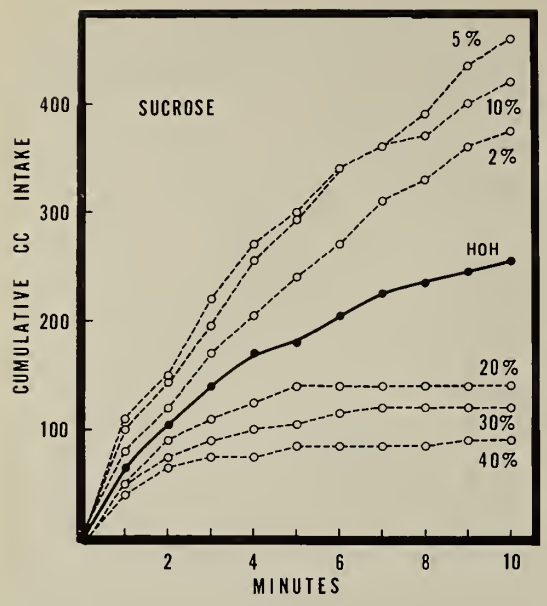


Figure 26–13. Cumulative intakes of different concentrations of sucrose of a human subject, showing preferences for weak concentrations and aversions to strong, hypertonic concentrations.

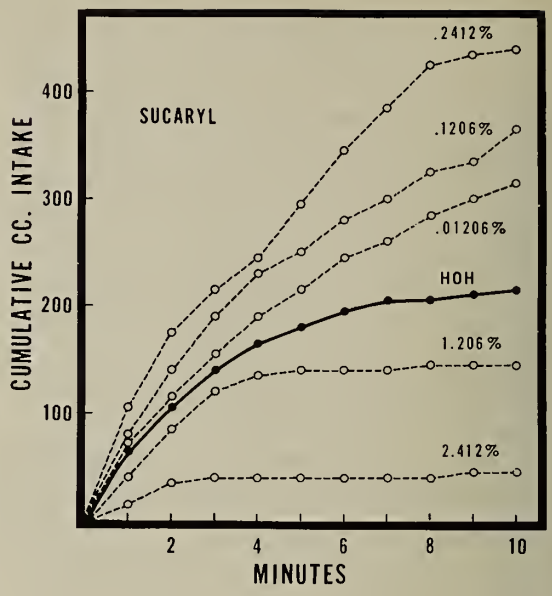


Figure 26–15. Cumulative intakes of different concentrations of sucaryl by a human subject, showing preference for the weak concentrations and aversion to the high, but not hypertonic, concentrations.

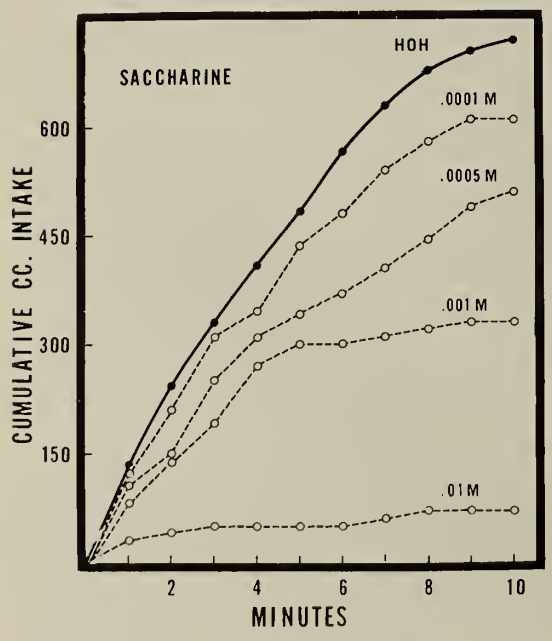


Figure 26–14. Cumulative intakes of different concentrations of saccharine by a human subject, showing aversion for all concentrations tested.

the aversion to hypertonic sucrose; "too sweet," "nauseatingly sweet." This suggests a mouth factor, possibly one dependent upon learning, since these concentrations are not hypertonic so there could not be that kind of postingestional effect.

As a result of these findings, we have become more directly concerned with the hedonic aspect of the oral and postingestional contributions to preference and aversion and have tried to duplicate some of the work of Cabanac.[1] Cabanac and his colleagues asked subjects to rate the "sweetness" and the "hedonic quality" of 50 ml mouthfuls of 20% glucose under two conditions. In one, the subject received the 50 ml of 20% glucose every 3 minutes, spat it out after 15 seconds, rinsed with water and rated pleasantness-unpleasantness on a +2

to −2 scale ("O" neutral) or a 5–1 scale (3.0 neutral). The second condition was the same except that the subject swallowed the 50 ml 20% glucose each trial, ingesting 1.5 liters in a 90–minute test of 30 trials. As might be expected, the judgment of sweetness did not change as testing progressed in either condition. Furthermore, in the first condition, the judgment of pleasantness did not change during the test. But when sugar was ingested in the second condition, the ratings reflected less and less pleasantness and more and more unpleasantness as the test proceeded (Fig. 26–16). A 500 ml gastric preload of 20% glucose before the test produced the same shift from pleasantness to unpleasantness in the ratings even in the condition where the glucose was spit out on each trial. But strangely enough, an intravenous load of glucose did not have any effect.

Using sophisticated observers in our laboratory as subjects we were able to replicate these findings fairly well. However, when we disguised the preloads of 0, 10, 20, 30, and 40% glucose by adding saccharine and yellow vegetable dye to them we ran into

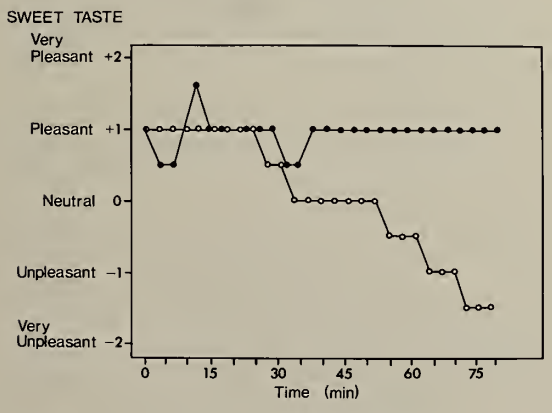


Figure 26–16. Rating of the pleasantness of sucrose by a human subject when each 50 ml mouthful sampled is spit out (closed circles) and when it is swallowed (open circles). After Cabanac.

inconsistencies. In some subjects the 40% preload had the biggest effect and in others the 10% or the water preload did, and the subjects could not "guess" which preload they got. Averaging the data for the four subjects submerged the inconsistencies and produced slightly increasing effects as a function of preload sugar concentration. However, the individual inconsistencies were bothersome, especially in the light of the fact that reproducing these phenomena in naive subjects has proved very difficult. Under a variety of testing conditions, naive subjects were always variable, sometimes showing an effect, sometimes not.

At this stage of our work, we believe the Cabanac phenomenon exists, but that it is not robust. Subjects feel slight nausea during these experiments and may be confusing their desire to ingest any more sugar with a change in the pleasantness of its taste. For example, when we told our subjects toward the end of a series of trials that they didn't have to ingest any more, but could spit, the ratings of the 20% glucose became more positive.

Furthermore, the distress or slight nausea the subjects reported may have been partly due to gastric distension *per se,* for in some cases, water in the stomach produced as strong a negative effect as sugar solution in the stomach. It may be for these reasons that intravenous glucose has no effect on the ratings of pleasantness. While our results are still inconclusive, Cabanac's method may yet prove to be a valuable approach to the central problem of unravelling the contributions of taste, postingestional factors, and internal environment state in the hedonic judgments of pleasantness that may accompany sweet preference.

CONCLUSIONS

1) *Preference varies as a function of the method of measurement and the condition of the organsim at the time of testing.* The major methods used in animal and human studies are: a) ingestion as in the single stimulus method,[7] b) choice with ingestion [10] or without as in the brief exposure method,[14] c) instrumental response reflecting willingness to work or motivation,[12] d) hedonic rating and subjective report in man.[1]

2) *Two sets of factors operate in preference and hedonic behavior: oral and postingestional factors.* The oral factors are principally taste, but may be mouth osmotic factors, and of course, texture and temperature. The postingestional factors may be gastric (e.g. gastric distension), osmotic, general dehydrational or nutritional state or may involve nausea.

3) *Learning most certainly plays a role in sweet preference, especially in its development.* Learning may play its role through taste as in Harriman's experiment [4] where adrenalectomized rats died because they refused NaCl solutions and drank sugar solution instead if they had an established sugar preference preoperatively. Or learning could function through postingestional effects (nausea) as it does when making a rat sick with LiCl after drinking a novel, sweet solution changes sweet preference to aversion.[9]

4) *All of these factors (the state of the organism, oral, postingestional, and learning factors) must be integrated in a common brain mechanism that controls a) regulation of ingestion, b) motivation, and c) hedonic rating of sweet substances.* These are the components of which sweet preference are composed and the final common path is in the brain as it is in other cases of regulation, motivation, and hedonic experience.[11]

REFERENCES

1. Cabanac, M.: Physiological role of pleasure. *Science,* 173: 1103–1107, 1971.
2. Dethier, V. G. and Rhoades, M. V.: Sugar preference-aversion functions for the blowfly. *Journal of Experimental Zoology,* 126: 177–204, 1954.
3. Hagstorm, E. C. and Pfaffmann, C.: The relative taste effectiveness of different sugars for the rat. *Journal of comparative and Physiological Psychology,* 52: 259–262, 1959.
4. Harriman, A. E.: The effect of a preoperative preference for sugar over salt upon compensatory salt selection by adrenalectomized rats. *Journal of Nutrition,* 57: 271–276, 1955.
5. Jordan, H. A., Wieland, W. F., Zebley, Susan P., Stellar, E. and Stunkard, A.J.: Direct measurement of food intake in man: A method for the objective study of eating behavior. *Psychosomatic Medicine,* 28: 836–843, 1966.
6. McCleary, R. A.: A physiological mechanism of sugar preference. Ph.D. Thesis, 1950–51, Johns Hopkins University.
7. McCleary, R. A.: Taste and postingestion factors in specific-hunger behavior. *Journal of comparative and Physiological Psychology,* 46: 411–421, 1953.
8. Mook, D.: Oral and postingestional determinants of the intake of various solutions in rats with esophageal fistulas. *Journal of comparative and Physiological Psychology,* 56: 645–659, 1963.

9 Nachman, M.: Learned taste and temperature aversions due to lithium chloride sickness after temporal delays. *Journal of comparative and Physiological Psychology,* 73: 22–30, 1970.

10. Richter, C.P. and Campbell, H.K.: Taste thresholds and taste preferences of rats for five common sugars. *Journal of Nutrition,* 20: 31–46, 1940.

11. Stellar, E.: Brain mechanisms in hunger and other hedonic experiences, *Proceedings of American Philosophical Society,* 118: 276–282, 1974.

12. Stellar, E.: Drive and motivation. In Magoun, H.W., *Section on Neurophysiology, Handbook of Physiology.* Washington, D.C., American Physiological Society, 1960, 1501–1527, see pp. 1520–1521.

13. Stellar, E.: Hunger in man: comparative and physiological studies. *American Psychologist,* 22: 105–117, 1967.

14. Young, P.T.: Studies of food preference, appetite, and dietary habit I. Running activity and dietary habit of the rat in relation to food preference. *Journal of comparative Psychology,* 37:327–370, 1944.

Chapter 27

SWEET ABUSE AND THE ADDICTION MODEL

John L. Falk

It has been estimated that in the United Kingdom between 1819 and 1930 sugar consumption per capita increased about 4-fold.[1] The significance of this increase should be viewed in the light of both animal-experimental and epidemiological data linking increased sugar consumption to arteriosclerotic and degenerative heart disease, obesity, hypertension and dental caries.[1] It is worth considering if there are general environmental factors which produce an overindulgence of substances such as various drugs, ethanol and sugar. Further, how overindulgence might relate to the notion of "addiction" should be evaluated, especially in relation to sweet substances.

WATER ABUSE

First, let us consider how a rather simple environmental manipulation leads to the abuse of, i.e., the massive overindulgence in, water. The abuse of this vehicle has never been a public health problem of any magnitude, but the production of polydipsia is nonetheless instructive and has provided us with a method for investigating the overindulgence of more pharmacologically significant substances, such as ethanol.

When a rat is gradually reduced to 80 or 90% of its free-feeding body weight by limiting its daily food ration and permitted to earn most of its food by lever-pressing on an intermittent food delivery schedule, a copious polydipsia develops concurrently with the consumption of the food pellets.[6] In a typical experiment, an animal will obtain a 45-mg food pellet on the average once per minute (a variable-interval one-minute schedule) for about 3 hours each day. When maintained in this fashion, animals drink almost 100 ml of water during the 3 hours, which is about one-half their body weight.[6] It is important to note that these aminals are never deprived of water and have continuous access to it in their home cages between the 3-hour food sessions.

A typical session is shown in Figure 27–1. Lever pressing is shown on the bottom channel and is not an important feature of the figure for our present purposes. The upper (cumulative) channel shows that bursts of licking occur immediately after the earning of most food pellets. Food pellet delivery is indicated by the reset

Acknowledgment: Supported by Grant AA 00253 from the National Institute of Alcohol Abuse and Alcoholism, Grant AM 14180 from NIH (Arthritis, Metabolism and Digestive Diseases), and Biological Sciences Support Grant (NIH) to Rutgers University.

of this channel or a vertical hatch mark if the pen is already reset (i.e., if the previous pellet consumption failed to be followed by a drinking burst). It can be seen that in most cases the animal takes a draught of water after consuming each food pellet. During such daily sessions, for months on end, the animal will continue to drink these massive amounts (about 3.4 times its pre-experimental, normal 24-hour water intake level); little water is drunk during the remaining 21 hours per day spent in the home cage. This pattern of excessive drinking (schedule-induced polydipsia) has been repeatedly confirmed not only in every strain of rat attempted but in mice, monkeys and chimpanzees (for general reviews see ref. 7,8). As there is no physiological state imposed by the experimental conditions which accounts for the overdrinking, i.e., there is no change in body water location or concentration, defect in renal concentrating ability, etc.,[7] the dipsogenic response can be referred to as "psychogenic polydipsia," a term used in clinical medicine to indicate massive overdrinking not explicable in terms of body water, hormonal, or renal changes.

ETHANOL ABUSE

Let us pass from the problem of water intake abuse to the oral abuse of ethanol. If animals are given continuous access in their home cages to ethanol solutions in various concentrations as the sole drinking fluid, a long history covering several decades of research, has failed to show any development of physical dependence on ethanol.[18] Furthermore, in experimental situations where ethanol solutions have been offered in choice situations paired with other fluids, no substantial and enduring increase in ethanol intake has been induced which has resulted in physical dependence by any experimental manipulation.[15] Given this rather discouraging history, but the undeniable fact of human oral ethanol overindulgence, we applied the schedule-induced polydipsia technique to the

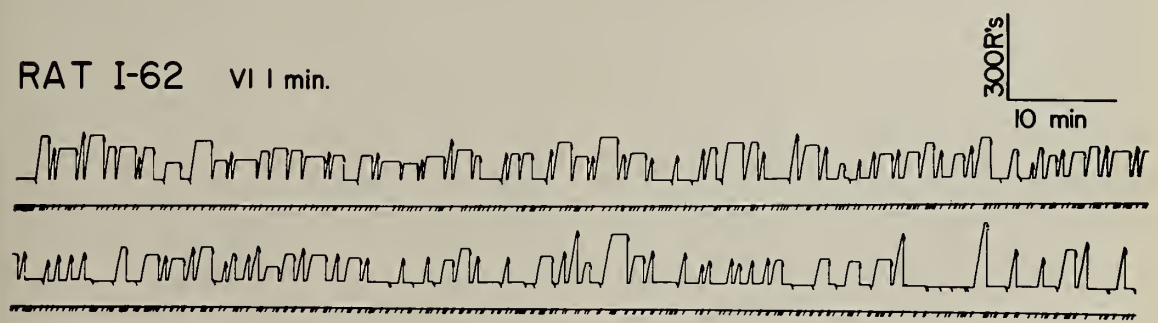


Figure 27–1. Drinking bursts on a variable-interval one-minute schedule (VI 1 min, 45-mg food pellets). Food pellet delivery resets upper channel to bottom or makes hatch mark. These cumulative responses are the drinking bursts. Lower channel shows lever pressing.

problem in the hope of producing oral overindulgence to the point of physical dependence. In our initial study,[9] we applied the technique in much the same way that gave us the water abuse result, but with a few important differences. Rats were reduced slowly to 80% of their free-feeding weights and then simply fed food pellets automatically, rather than being required to earn them by lever-pressing, on a 24-hour schedule in individual cages. Every 2 minutes a 45-mg food pellet was delivered during 1-hour feeding periods that were separated by 3-hour intervals. Thus, there were 6 feeding periods in each 24-hour cycle, delivering a total of 180 pellets. First, water was the available fluid and after schedule-induced polydipsia was established, a gradually increasing concentration of ethanol was substituted in place of water. Starting with 1% (by volume) ethanol, this concentration was increased every 6 to 8 days in 1% increments to 6% ethanol.

Figure 27–2 shows the amount of ethanol ingested by each of 8 rats as a function of the concentration offered. Except for rat No. 6, animals ingested between 11 and 15 g ethanol per kg of body weight at 6% ethanol. The change from 5 to 6% ethanol did not lead to an increased consumption of g ethanol/kg. Therefore, the concentration was returned to 5% for the ensuing 3 months, after which time the experiment was terminated with a test for the presence of physical dependence. The 180 food pellets per day plus the calories in the ingested ethanol permitted the body weights to increase continuously. At the end of the experiment, the weights had returned to the initial free-feeding

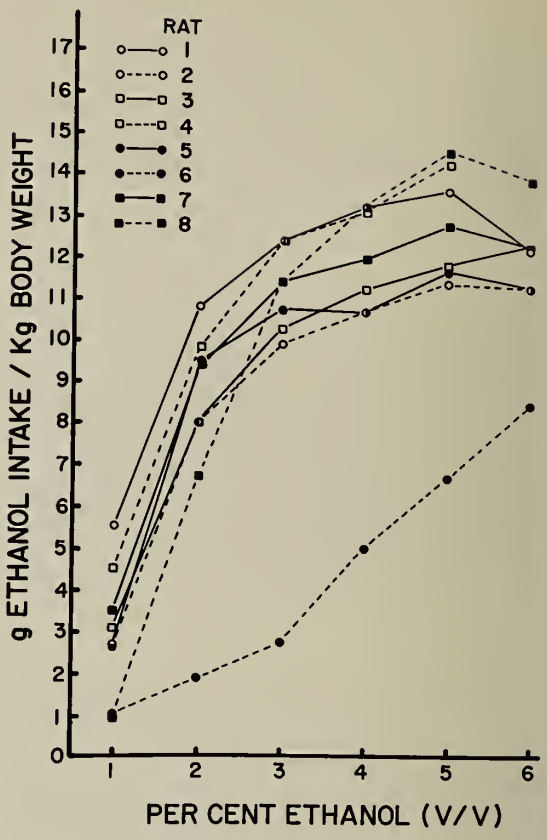


Figure 27–2. Mean daily amounts of ethanol drunk by individual rats as a function of the available ethanol concentration.

weight level when the test for physical dependence was given.

During the second month, we measured blood ethanol levels in samples from the tail taken one hour before and one hour after each of the 1-hour feeding periods (Fig. 27–3). The samples were taken serially from 8 am to 7 pm. Two weeks later the samples between 8 pm and 7 am were gathered. Thus, there is a data break indicated between 7 and 8 pm in the figure to denote the two-week interim. For most of the animals the blood ethanol concentration remained greater than 100 mg/100 ml for most of the time (6 pm to 11 am) and often lay between

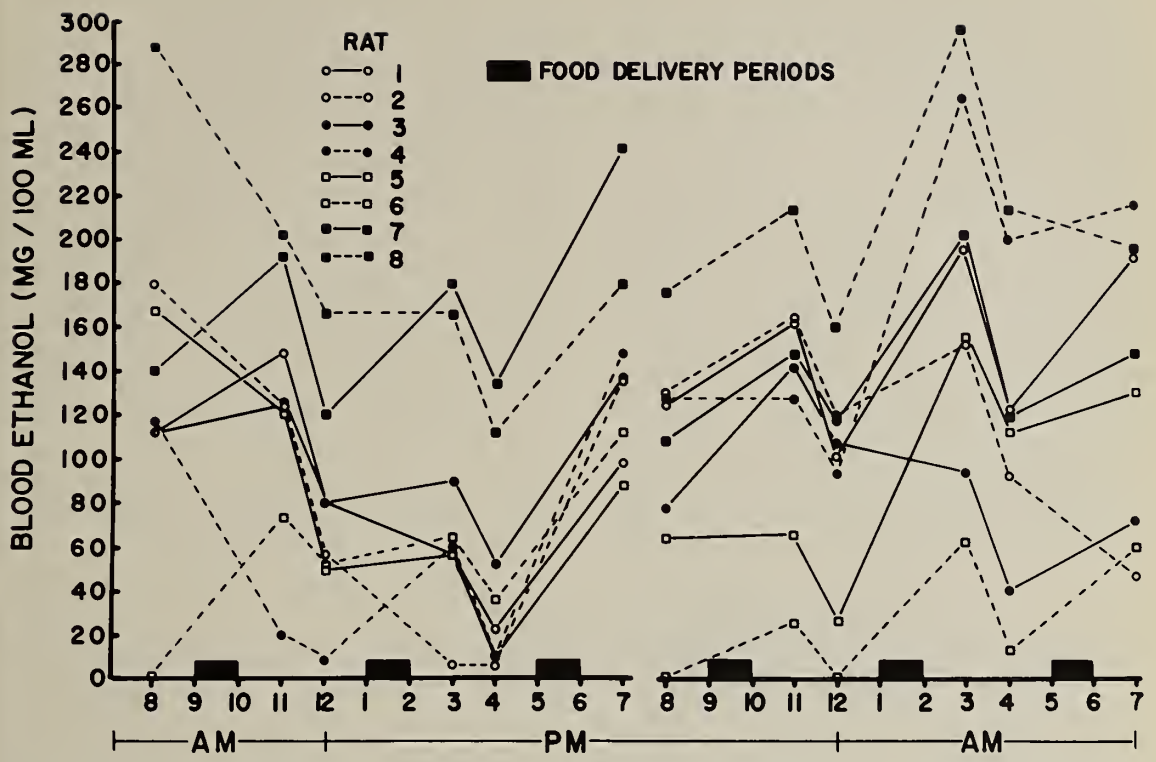


Figure 27–3. Concentrations of ethanol in the blood of rats drinking 5% ethanol. During the indicated 1-hour feeding periods, a food pellet (45 mg) was delivered every 2 minutes.

150 and 300 mg per cent. Daily intake of ethanol remained quite constant during the 3-month period on 5% ethanol. For the last 10 days of the experiment, the mean daily intake for the eight animals was 13.1 g ethanol/kg.

When these and similarly-treated animals in subsequent experiments [21] were withdrawn from ethanol for 8 hours and then subjected to a brief (1–2 seconds) stimulus consisting of a bunch of keys shaken over them in individual withdrawal cages, severe tonic-clonic seizures resulted, with some animals dying. Prolonged key shaking failed to induce either seizures or preconvulsive activity in control animals, in animals polydipsic on water held to 80% body weight, or rats held to 80% body weight with free access to 5% ethanol as their sole drinking fluid in a home-cage situation.[9,10] With this method then, we were able to produce unequivocal physical dependence on ethanol by oral self-administration of an aqueous ethanol solution in animals.

ETHANOL ABUSE AND COMPETING SWEET FLUIDS

In the above experiment, no choice was offered between 5% ethanol and any other fluid. Our aim had only been to produce oral overindulgence in ethanol for the production of chronic physical dependence. But with the demonstration that this chronic, stable state can be produced

in animals gaining weight and seemingly in good health, it was of interest to determine if such animals had an altered preference for the ethanol solution vis à vis other fluids. Accordingly, an experiment similar to the one described above was instituted except that 5% ethanol was available initially from each of two drinking tubes.[20] After establishing 5% ethanol polydipsia for 12 days (see Fig. 27–4, early EtOH only), a 3-day preference test was run between 5% ethanol in one tube and water in the other tube (Fig. 27–4, early H_2O vs EtOH). Tube positions were switched daily. This test, as well as a similar one run on days 76–78, indicated that the 5% ethanol was preferred to water. Following this determination, a series of preference tests between 5% ethanol and increasing concentrations of dextrose were run, commencing with 0.7% (w/v) dextrose. If an animal switched its preference from ethanol to a dextrose solution, the preference test was continued for at least 5 days after the switch occurred. Preference for a particular solution was defined as that solution whose intake level was more than 50% of the total fluid intake. After any animal's preference

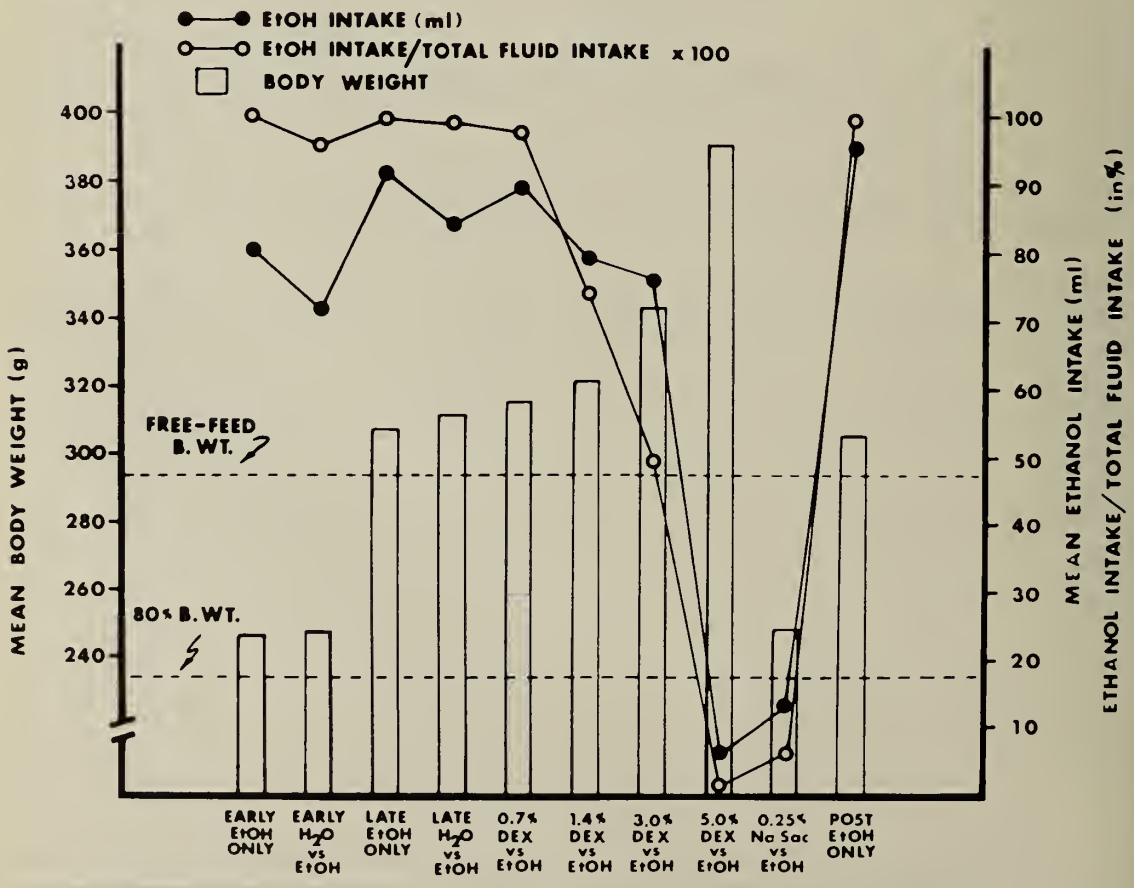


Figure 27–4. Preference relation between ethanol and other fluids (schedule-induced condition). All values are means.

switched from the ethanol solution to a dextrose solution (by the above criteria), a 0.25% sodium saccharin solution (w/v) was substituted for the dextrose (0.25 Na Sac vs EtOH). The saccharin was paired with the ethanol until the animal either switched back to ethanol (which never happened), or lost so much body weight that general health was compromised. At that point, 5% ethanol was again placed in both tubes and the ethanol drinking levels were redetermined (post EtOH only).

It is evident from Figure 27–4 that besides preferring 5% ethanol to water, when the dextrose concentration reached a high enough value, animals switched their fluid preference from ethanol to dextrose. Out of 8 rats, one switched at 1.4% dextrose, 2 rats at 3% dextrose, and the remaining animals at 5% dextrose. All animals preferred the noncaloric saccharin solution even after their weights had declined seriously. Two rats actually died in a state of anorexia, ceasing to eat their scheduled food pellets during the last few days but continuing to drink the saccharin solution. Thus, even with a caloric deficit which progressively developed over a 6-week period, all animals continued to prefer saccharin solution in the presence of a previously preferred ethanol solution which could have provided the calories necessary for body weight maintenance. Similar results were obtained with an additional group of animals which were treated similarly but were given no dextrose solution tests. Thus, the saccharin preference was not a result of any carry over of a preference for a sweet dextrose solution to a sweet saccharin solution.

Five out of the above 8 rats switched their preference from 5% ethanol to dextrose only when the dextrose solution concentration was increased to 5%. This result was compared to results from a group of animals that were maintained on similar fluid choices but not fed under the intermittent food regimen and thus did not have the polydipsic drinking pattern maintaining a continuously elevated blood ethanol level resulting in physical dependence.[20] They did, however, drink ethanol for the same period as the above group, functioning thus as a long-term adaptation to ethanol solution control group. Some animals were allowed to increase their body weights similarly to the above group while others were held to the 80% level. All animals were preferring dextrose at the 3% dextrose versus 5% ethanol choice phase of the testing (see Figs. 27–5 and 6). They also preferred the saccharin solution to the ethanol. Comparing Fig. 27–4 with 5 and 6, not only was the preference change point definitely reached at 5% dextrose compared to 3% dextrose, but the schedule-induced, dependent animals still drank a large volume of 5% ethanol during the 3% dextrose choice, while the other animals had decreased their intake of ethanol considerably at that comparison point. Although the schedule-induced ethanol dependent animals were more resistent than controls to the sweet blandishments of dextrose, they nevertheless switched when the concentration was raised sufficiently. No group, including the schedule-induced group which was switched directly from a test between 5% ethanol and water to a test between the ethanol and saccharin solutions, withstood the inducement of the sweet

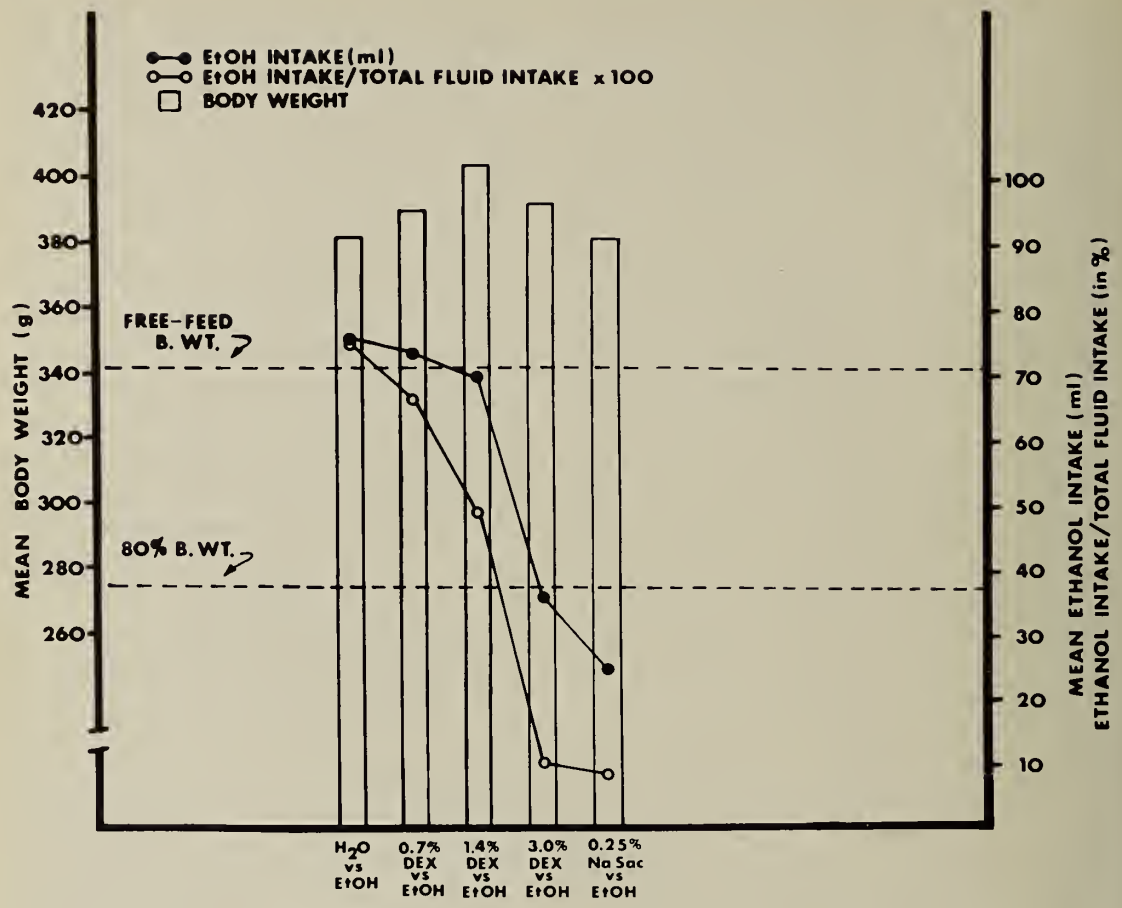


Figure 27–5. Preference relation between ethanol and other fluids. Single food-ration regimen with body weights allowed to increase. All values are means.

saccharin. Whether physically dependent on ethanol or not, this last preference change was a disasterous one for all animals in terms of caloric maintenance.

ETHANOL ABUSE AND SWEET TASTE: CONDITIONAL ENHANCEMENT

While increased intakes of ethanol have been reported when ethanol was mixed with various flavored solutions, these increases have not been sufficient to produce physical dependence.[18] Since 0.25% saccharin functioned as such a powerful determinant of choice and intake level in the previous experiment, we wished to ascertain whether the addition of saccharin to our standard ethanol solution would result in a further elevated level of ethanol intake. Accordingly, two groups of rats living in standard cages were maintained at 80% body weight and allowed free access to either 5% ethanol or a combined 5% ethanol—0.25% saccharin solution

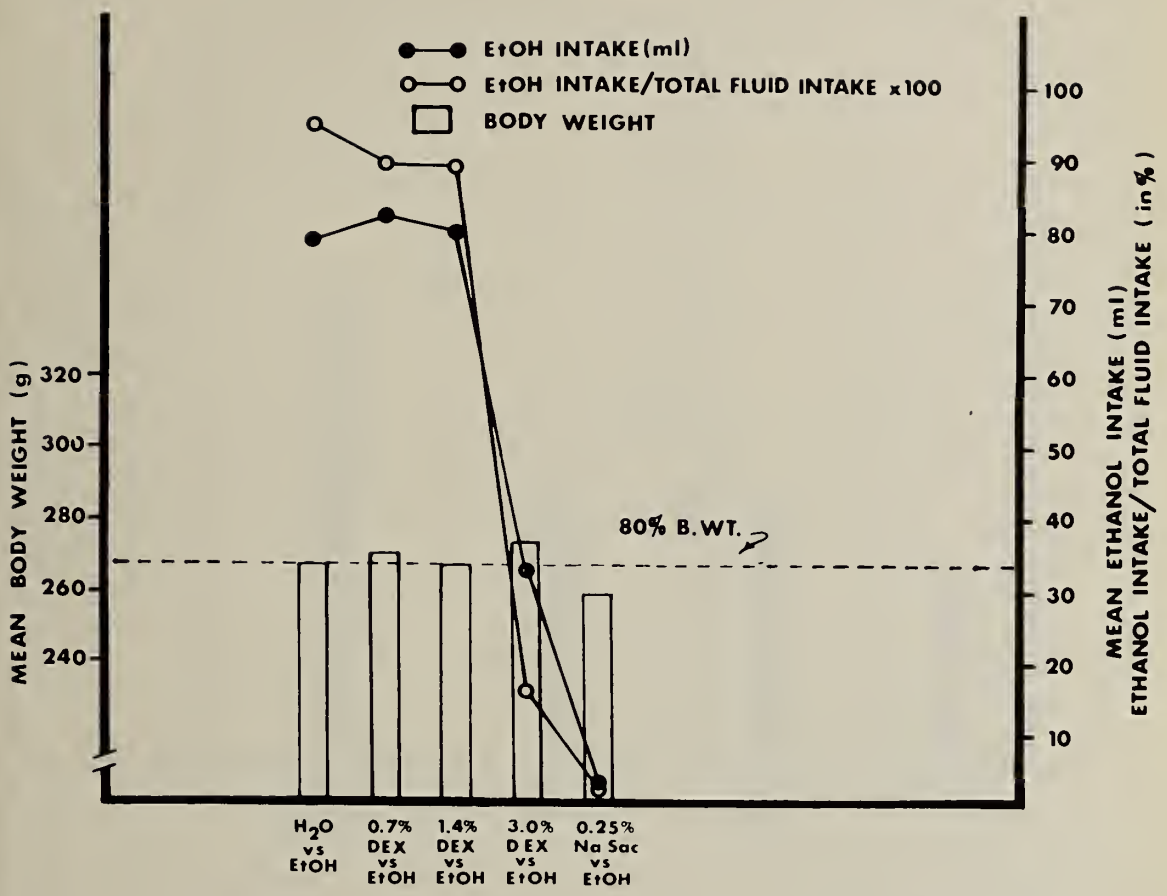


Figure 27–6. Preference relation between ethanol and other fluids. Single food-ration regimen with body weights maintained at 80%. All values are means.

for 3 months. Another group of animals was maintained on the schedule-induced polydipsia regimen previously described.[21] After one month on 5% ethanol, they were switched to the ethanol-saccharin solution as their drinking fluid for two months. As shown in Figure 27–7, there was no significant difference in the g ethanol/kg intakes between the two home-cage conditions. However, the addition of saccharin to the ethanol in the schedule-induced condition produced a marked increase in ethanol intake from 13.1 to 15.1 g ethanol/kg. The addition of saccharin in the schedule-induced condition enhanced ethanol intake but failed to have this effect in the non-schedule-controlled, home-cage condition. Thus, the gustatory component only worked in synergy with the schedule-induced condition which already produced an intake level greater than that of the home-cage condition.

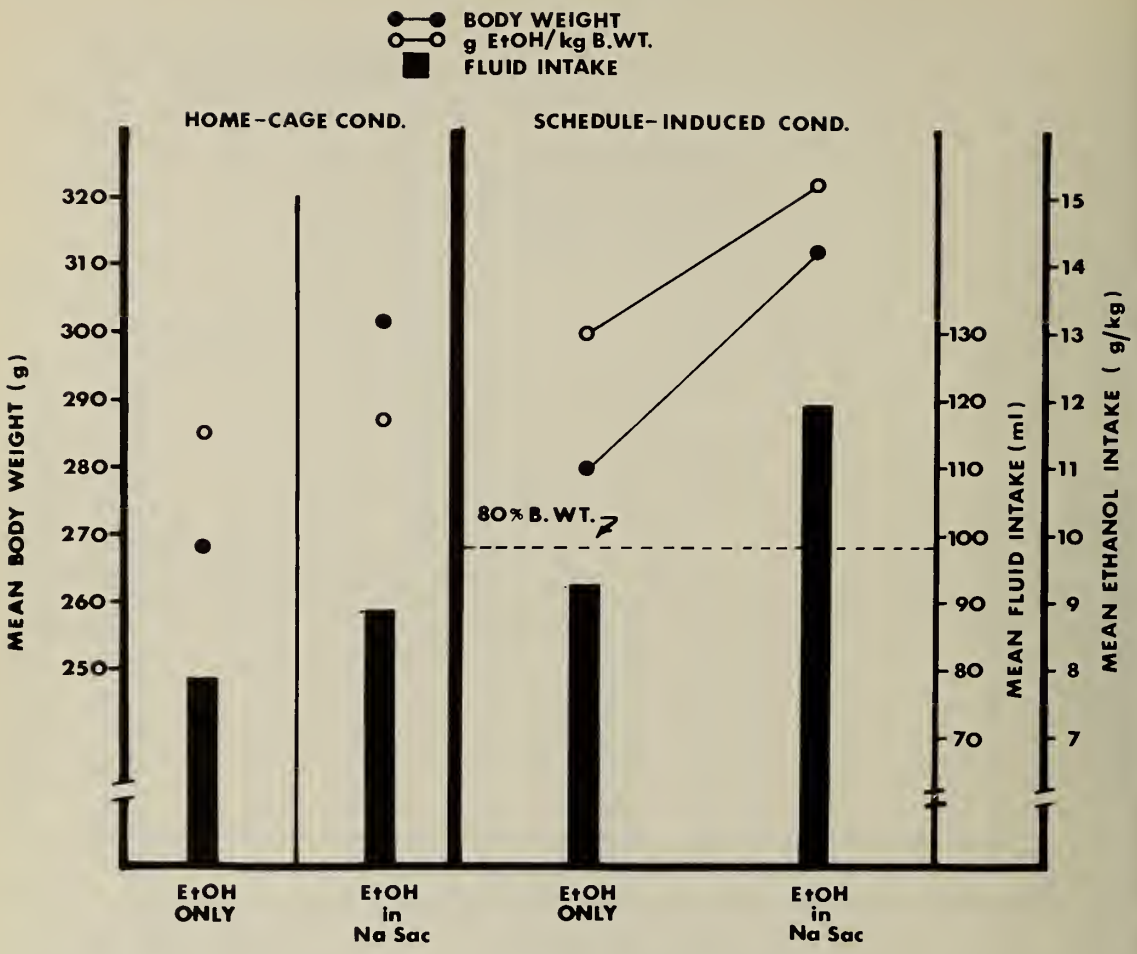


Figure 27–7. Effect of addition of sodium saccharin to 5% ethanol on intake in both home-cage and schedule-induced conditions. All data are means for the last 30 days in each condition. The body weights for the two home-cage conditions are the mean 80% weight levels.

ABUSE AND ADDICTION

Given an appropriate environmental condition (schedule-induced situation), a sweet substance, such as saccharin, can be used to produce an enduring and physiologically significant change in enhancing ethanol intake. Also, we have shown that both dextrose and saccharin solutions can function as alternatives to ethanol intake in physically dependent animals given an enticing enough concentration of the sweet alternative. Ethanol is the only substance to which there is widespread, fluid oral addiction. Nevertheless, appropriate sweet fluids can compete successfully with this addictive intake level in the dependent rat. This successful competition occurs not because sweet substances are pharmacological substitutes for ethanol (cross-tolerant or cross-dependent), but because such sub-

stances better satisfy the conditions for the production of an oral reinforcing agent, at least for the rat. There is no reason to believe that we are dealing with anything more mysterious or physiologically compelling than a reinforcement-efficacy hierarchy. In physically dependent animals, 5% ethanol successfully competes with 3% dextrose, but not in the non-dependent (home-cage) animals. But despite that dependent state, 5% ethanol cannot compete with 5% dextrose. Nor can it compete with 0.25% saccharin. In these cases the sweet fluid wins out and the animals withdraw from their ethanol preference. In so doing, they risk withdrawal convulsions, sacrifice considerable calories and, in the case of a persistent saccharin choice, even forfeit their lives. Since these sweets replace ethanol overindulgence in dependent animals and form a new and rather non-beneficial indulgence, it is reasonable to ask if these new choices are not themselves some form of addiction. Also, the synergistic effect that saccharin addition exerts upon ethanol overindulgence produced by schedule induction, and which fails to occur in the home-cage environment, lends weight to the notion that it is not just the chemical properties of ethanol or sweet fluids that define their reinforcing properties, but that the inducing environment plays an intimate role in determining these properties. If we are to argue that sweet and ethanol abuses are not radically divergent phenomena, then it should be demonstrable that the reinforcing efficacy of ethanol, like the sweets, has powerful situational components. This was shown by (a) the chronic over-indulgence of ethanol and the ensuing dependence induced by the food schedule (increasing the efficacy of ethanol), and (b) the abandonment of this chronic overindulgence and its associated dependence state in favor of a 5% dextrose alternative (decreasing the efficacy of ethanol).

Now it might be argued in the case of water and sweet abuses that the damages incurred are rather indirect. It takes unusual environmental arrangements for these substances to be abused and to become hazards to health, while ethanol and other agents with addiction liability produce their effects much more directly. The position is that such agents when chronically self-administered result in deleterious effects as a function of their pharmacological action either directly, or indirectly as a consequence of withdrawal (abstinence syndrome). Further, addictive agents are viewed as instituting biochemical changes in the central nervous system which, functioning in a vicious circle, maintain a craving for the particular agent. However, an alternate view is that there is no useful distinction to be drawn between abuse and addiction. Both imply overindulgence to a point where various other normally reinforcing events are displaced. Sources of this overindulgence are multiple and involve social determinants, as well as such accepted factors as direct (immediate pharmacological) efficacy and physical dependence. For example, the physiological compulsion or biochemical craving model has dominated both the popular and scientific views of alcoholism for several decades. However, recent research has indicated that moderate or controlled drinking in alcoholics does not trigger

irresistable "craving" or a "loss of control." [2,5,11,12,16,17] In addition, work on specific environmental manipulations which exert control over the drinking of chronic alcoholics has shown that when 10 or 15 minutes of physical and social isolation was the consequence of each drink, alcohol intake decreased about 50 per cent.[3] When 40 minutes of time-out from social interactions was the consequence of each drink, drinking was suppressed to very low levels when alternate sources of reinforcement (television, games, etc.) were not available.[13] The restriction of these other privileges as a general living condition itself had no systematic effect on the level of drinking. But without access to such alternatives, the contingent social time-outs consequent upon each drink markedly suppressed drinking. These results are strong evidence against a biochemically-driven craving notion as the underlying substrate maintaining the drinking of the alcoholic. With the contingent withdrawal of social interaction, drinking is not supported.

The physiological and supposed biochemical bases of addiction have been emphasized in the past, but the above studies indicate that alcoholic overindulgence is not explicable in terms of a run-away biochemical state. Controlled, moderate drinking by "alcoholics" is feasible [2,5,11,12,16] and priming alcoholics with ethanol fails to result in any onset of craving.[17] The metabolic hypothesis as an explanation of the continued abuse of narcotics has also been questioned.[4]

It is clear, then, that overindulgence in various commodities can have bases which are not solely attributable to biochemical predispositions or the development of physical dependence. The environmental sources of the water abuse (schedule-induced polydipsia) previously described reveal one sort of prototype situation; the environmental factors instituting, maintaining and suppressing ethanol abuse in animals and humans yield further insights.

SWEET ABUSE

There are epidemiological and physiological studies indicating the increased use of sugars and their biomedical hazards.[1] One interesting line of animal research has shown that if rats are allowed one hour per day to ingest food they can regulate body weight on this regimen. However, if a sucrose solution is available concurrently with the food, rapid weight loss and even death occur in about 3 weeks.[14,19] Animals drink the sucrose solution and fail to eat enough food in the hour to sustain life. Such a situation emphasizes the continuity between overindulgence and addiction. A life-sustaining reinforcer (food) is displaced by chronic sucrose overindulgence.

It is not a coincidence that commodities overindulged in are also ones that function as immediate reinforcers, e.g., morphine, cocaine and sweets. Morphine is self-administered by monkeys without a prior physical dependence being established and at low dose levels where dependence cannot be shown to develop.[22] Physical dependence, then, does not explain the initial reinforcing properties

of drugs such as the opioids and cocaine, their chronic use, nor their strong re-addiction liability. Such drugs, as well as sweets, have immediate reinforcing properties, but this alone is probably not sufficient for the production of overindulgence. Special environmental arrangements will permit or produce overindulgence of these substances and even of water. Such arrangements include schedule-induction, concurrent food and sucrose solution presentation on a limited feeding opportunity regimen, and the non-withdrawal of social interactions as a consequence of ethanol drinking. Sweet substances have immediate reinforcing properties to many animals. They are highly preferred and accepted in large volumes under various regimens. For humans, sweets are provided in convenient solid and liquid packages suitable to environmental conditions allowing only intermittent, "convenient," and unceremonious feeding. The repetitive consummatory responses evoked by sweetened convenience foods in such time-limited, intermittent feeding situations may become the individual's characteristic feeding pattern. The ubiquity of sweet additives to a wide range of items in the diet (breakfast foods, beverages, "chinese" food, etc.) could function as a stimulus factor determining the above feeding pattern. Sweet may often function as a hidden condiment acquiring additional conditioned reinforcing properties owing to its association with proteins in the modern diet.

Such speculations aside, is sweet abuse an addiction? There is no evidence of dose escalation (tolerance) nor are there withdrawal symptoms which are not simply attributable to caloric deprivation. But evidence has been presented indicating that the phenomena of tolerance and physical dependence explain little about the initiation and maintenance of ethanol and other drug overindulgence. The factors determining all these overindulgences may have to be sought in environmental conditions which synergize the intake of substances already possessing immediate-reinforcer properties.

REFERENCES

1. Ahrens, R. A.: Sucrose, hypertension, and heart disease: an historical perspective. *Amer. J. Clin. Nutrit.* 27: 403–422, 1974.
2. Bigelow, G., Cohen, M., Liebson, I. and Faillace, L. A.: Abstinence or moderation? Choice by alcoholics. *Behav. Res. Ther.* 10: 209–214, 1972.
3. Bigelow, G., Liebson, I. and Griffiths, R.: Alcoholic drinking: suppression by a brief time-out procedure. *Behav. Res. Ther.* 12: 107–115, 1974.
4. Bowden, C. L. and Maddux, J. F.: Methadone maintenance: myth and reality. *Amer. J. Psychiat.* 129: 435–440, 1972.
5. Cohen, M., Liebson, I. A., Faillace, L. A. and Allen, R. P.: Moderate drinking by chronic alcoholics: a schedule-dependent phenomenon. *J. nerv. ment. Dis.* 153: 434–444, 1971.
6. Falk, J.L.: Production of polydipsia in normal rats by an intermittent food schedule. *Science* 133: 195–196, 1961.
7. Falk, J.L.: Conditions producing psychogenic polydipsia in animals. *Ann. N.Y. Acad. Sci.* 157: 569–593, 1969.
8. Falk, J.L.: The nature and determinants of adjunctive behavior. *Physiol. Behav.* 6: 577–588, 1971.
9. Falk, J. L., Samson, H. H. and Winger, G.: Behavioral maintenance of high

concentrations of blood ethanol and physical dependence in the rat. *Science* 177: 811–813, 1972.

10. Falk, J. L., Samson, H. H. and Winger, G.: Behavioral maintenance of high concentrations of blood ethanol and physical dependence in the rat. *Science* 180: 881, 1973.
11. Gottheil, E., Alterman, A. I., Skoloda, T. E. and Murphy, B. F.: Alcoholics' patterns of controlled drinking. *Amer. J. Psychiat.* 130: 418–422, 1973.
12. Gottheil, E., Crawford, H. D. and Cornelison, F. S.: The alcoholic's ability to resist available alcohol. *Dis. Nerv. System* 34: 80–82, 1973.
13. Griffiths, R., Bigelow, G. and Liebson, I.: Suppression of ethanol self-administration in alcoholics by contingent timeout from social interactions. *Behav. Res. Ther.* 12: 327–334, 1974.
14. Hamilton, L. W.: Starvation induced by sucrose ingestion in the rat: partial protection by septal lesions. *J. comp. physiol. Psychol.* 77: 59–69, 1971.
15. Mello, N.K.: A review of methods to induce alcohol addiction in animals. *Pharmac. Biochem. Behav.* 1: 80–102, 1973.
16. Mello, N.K. and Mendelson, J. H.: Experimentally induced intoxication in alcoholics: a comparison between programmed and spontaneous drinking. *J. Pharmac. exp. Ther.* 173: 101–116, 1970.
17. Merry, J.: The "loss of control" myth. *Lancet* 1: 1257–1258, 1966.
18. Myers, R. D. and Veal, W. L.: The determinants of alcohol preference in animals. In Kissin, B. and Begleiter, H. (Eds.), *The Biology of Alcoholism,* New York, Plenum, 1972, pp. 131–168.
19. Navarick, D. J.: Effect of saccharin and sucrose consumption on adjustment to a food deprivation schedule. Master's thesis, Rutgers University, 1969.
20. Samson, H. H. and Falk, J. L.: Alteration of fluid preference in ethanol dependent rats. *J. Pharmac. exp. Ther.* 190: 365–376, 1974.
21. Samson, H. H. and Falk, J. L.: Schedule-induced ethanol polydipsia: enhancement by saccharin. *Pharmac. Biochem. Behav.* 2: 835–838, 1974.
22. Schuster, C. R. and Thompson, T.: Self-administration of and behavioral dependence on drugs. *Ann. Rev. Pharmac.* 9: 483–502, 1969.

Chapter 28

SIX COMMON SUGARS AS TOOLS FOR THE STUDY OF APPETITE FOR SUGAR

Curt P. Richter

Observations made on the appetite shown by rats for a single sugar, for instance glucose, do not throw any light on the origin of the sugar appetite. We do not know whether the amount of the sugar eaten by the rat is a function of solubility of the sugar or of its sweet taste or its absorption rate, etc.

I bring this up because a combined study of the six common sugars—glucose, sucrose, fructose, maltose, lactose, galactose—makes it possible to determine significance of the intake of each one of the sugars. The study brings out facts that could not be obtained, even with the most extensive study with a single sugar. Use of the six sugars for this purpose depends on the fact that they have a number of very different physical and physiological characteristics; and that each sugar may be ranked for each characteristic. Some of the results were presented in earlier reports.[7,9]

Table 28–I lists these characteristics and shows the ranking of each of the six sugars. Thus for molecular weight—glucose and galactose rank at the top, being the smallest and maltose and lactose at the bottom; for solubility in water—fructose and sucrose rank at the top, next maltose and glucose, next lactose and galactose at the bottom; for sweetness—fructose and sucrose again are at the top and galactose and lactose are at the bottom; for absorption rate—galactose is at the top and fructose at the bottom; for production of alimentary hyperglycemia—galactose and maltose are at the top and lactose and fructose at the bottom; and finally for limits of assimilability—maltose and glucose are at the top, lactose and galactose are at the bottom.

Comparison of rankings of maximum intakes of each of the six sugars with rankings of the properties of the sugars in Table 28–I should throw light on factors affecting sugar appetite.

METHODS

Methods used in determining maximum intakes of the six sugars have been described.[7] The so-called taste preference technique was used. Each rat kept in a small rectangular wire mesh cage had access to a food

Acknowledgment: This work has been carried out under a grant from the National Institute of Mental Health (MH00576).

TABLE 28–I

RANKING OF SIX COMMON SUGARS IN RELATION TO VARIOUS PROPERTIES

Molecular Weight		*Solubilities in Water*	*Sweetness*	*Absorption Rate*	*Production of Alimentary Hyperglycemia*	*Limits of Assimilability*
			Biester, Wood and Wahlin (1925)	*Cori (1925)*	*Rheingold and Karr (1927)*	*Ariyama and Takahasi (1929)*
			Man	*Rat*	*Rabbit*	*Rat*
GLUCOSE	180.16	FRUCTOSE	FRUCTOSE	GALACTOSE	GALACTOSE	MALTOSE
GALACTOSE	180.16	SUCROSE	SUCROSE	GLUCOSE	MALTOSE	GLUCOSE
FRUCTOSE	180.16	MALTOSE	GLUCOSE	FRUCTOSE	SUCROSE	SUCROSE
SUCROSE	342.30	GLUCOSE	MALTOSE		LACTOSE	FRUCTOSE
MALTOSE	360.31	LACTOSE	GALACTOSE		FRUCTOSE	LACTOSE
LACTOSE	360.31	GALACTOSE	LACTOSE			GALACTOSE

cup (stock diet *) and two graduated inverted 100 milliliter water bottles. The bottles were made as nearly alike as possible and were placed in strictly symmetrical positions in the cage to make certain that the containers or position did not affect the rat's choice of sugar. (The rat made its selections only on the basis of taste). For 10–15 days both bottles were filled with distilled water. Then the bottle from which the rat drank smaller amounts was filled with a subliminal concentration of the sugar to be tested. Concentrations of the sugar were then increased by small amounts each day until the rat increased its intake of the sugar and decreased its intake of distilled water. This concentration was taken as the taste threshold. As the concentrations were increased still higher the rats drank progressively more of the sugar solution and less of the water until a peak intake was reached beyond which they decreased their daily intake of the sugar solution but still maintained their high intake of sugar.

All of the sugars were highly purified grade. Sucrose (U.S.P.), glucose (U.S.P.), lactose (U.S.P.), maltose (C.P.), galactose (C.P.), fructose (C.P.).

To demonstrate effects produced on sugar appetites by increased metabolic rates parallel observations were made on hyperthyroid rats (0.2, 0.1, 0.15 per cent dessicated thyroid powder mixed with stock diet).

RESULTS

Taste thresholds

Table 28–II shows the taste threshold records for 54 normal rats and 26 rats made hyperthyroid by feeding of thyroid powder. The table shows that maltose had the lowest and most consistent thresholds. The wide range of variations in thresholds for fructose made it useless to obtain averages. All of the rats preferred maltose, glucose, sucrose, fructose, and galactose to water but none preferred lactose to water. These differences are illustrated by the two charts in Figure 28–1. The chart at the left shows the clear cut distinction that eight rats made between glucose and distilled water. The other chart shows that at no concentration did nine rats prefer lactose to water. Up to a 4 per cent lactose concentration the animals showed no preference for lactose but above that they began to refuse it.

*Graham flour–72.5%, skimmed milk powder–10.0%, casein–10.0%, butter–5.0%, calcium carbonate–1.5%, sodium chloride–1.0%.

Maximum intake levels of the six sugars

The two graphs in Figures 28–2 A & B show how the maximum intakes of sugars were obtained for normal and hyperthyroid rats. "A" shows that glucose was offered to a normal rat in concentrations that were increased daily up to 30 per cent. The intake of the sugar solutions reached a peak at 11 per cent and then decreased at a steady rate to 30 per cent. However, with increasing concentrations, intake in weight of glucose remained about the same as at the peak. "B" shows that at the peak concentration of glucose (9 per cent) this hyperthyroid rat drank about twice the amount taken by the normal rat.

The grams of sugar per kilogram

TABLE 28–II

INDIVIDUAL TASTE PREFERENCE THRESHOLDS OF 54 NORMAL AND 26 HYPERTHYROID RATS FOR SIX COMMON SUGARS IN PERCENTAGE CONCENTRATION

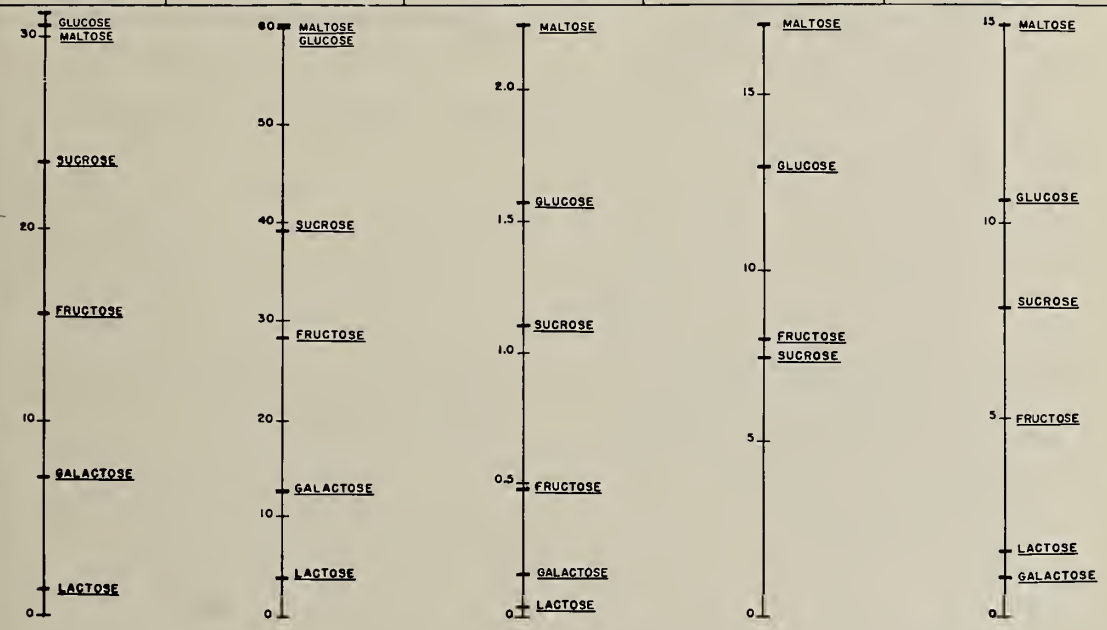
MALTOSE		*GLUCOSE*		*SUCROSE*		*FRUCTOSE*		*GALACTOSE*		*LACTOSE*	
Normal	*Hyper-thyroid*	*Normal*	*Hyper-thyroid*	*Normal*	*Hyper-thyroid*	*Normal*	*Hyper-thyroid*	*Normal*	*Hyper-thyroid*	*Normal (rejection)*	*Hyper-thyroid (acceptance)*
0.04	0.04	0.1	0.1	0.1	0.08	0.1	0.4	0.5	1.0	2.8	0.8
0.04	0.06	0.2	0.5	0.4	0.4	0.2	0.7	0.8	1.2	3.0	2.0
0.06	0.06	0.2	0.7	0.4	0.7	0.2	3.0	1.0	1.8	4.2	
0.06	0.08	0.2	0.7	0.4	0.8	0.2	3.0	1.2	1.8	4.6	
0.06		0.3	0.8	0.6		0.2		1.2		4.6	
0.06		0.3	0.8	0.6		0.2		1.2		5.0	
0.08		0.3	1.6	0.8		0.7		1.8		6.0	
0.08		0.4	3.0			3.0		1.8		6.5	
0.08						3.0		2.4		6.5	
0.08						5.0		2.8			
Mean concentration 0.064	0.060	0.25	1.03	0.50	0.50	a		1.47	1.45	4.80	
Standard deviation 0.005	0.008	0.03	0.32	0.10	0.16			0.23	0.21	0.46	

Note a: Mean and standard deviation not calculated due to markedly non-normal distribution.

TABLE 28–III

RANKING OF THE APPETITES SHOWN BY NORMAL AND HYPERTHYROID RATS FOR SIX COMMON SUGARS AND THE LIMITS OF ASSIMILABILITY OF THESE SUGARS

AVERAGE DAILY INTAKE OF SUGARS IN GM/K		RATIO SUGAR CAL./K FOOD CAL./K	LIMITS OF ASSIMILABILITY GM/K	
CONCENTRATIONS - 9% - 30% INCLUSIVE			MENDEL AND JONES (1920) RABBITS	ARIYAMA AND TAKAHASI (1929) NORMAL RATS
NORMAL RATS	HYPERTHYROID RATS	HYPERTHYROID RATS		



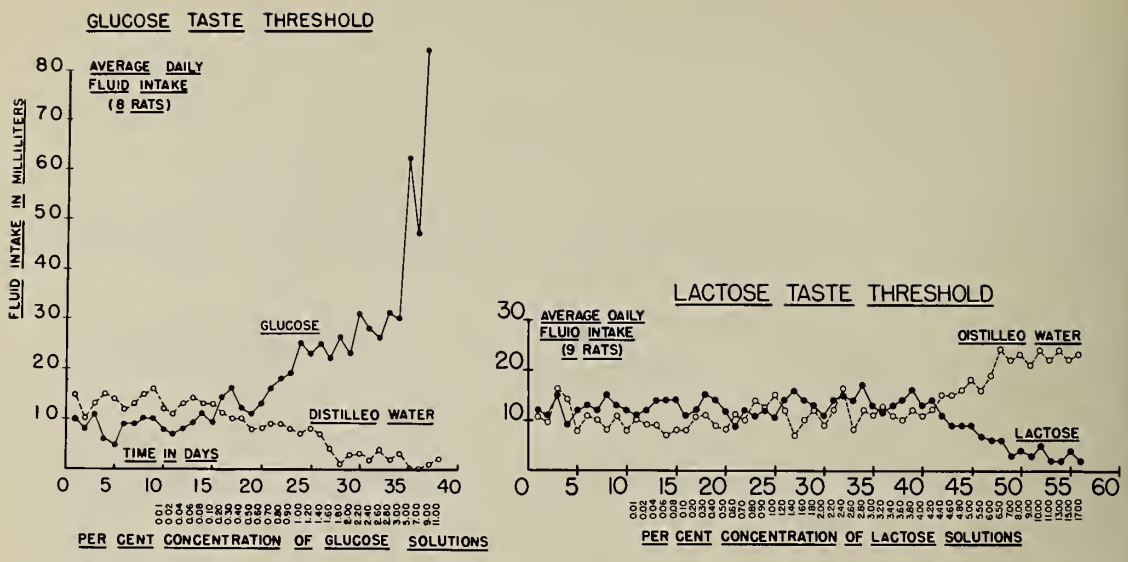


Figure 28–1 Graphs showing average taste thresholds for eight rats on glucose and for nine rats on lactose.

body weight obtained at various concentrations by normal rats "A" and hyperthyroid rats "B" are shown in Figure 28–3. Normal rats ingested maltose and glucose in the highest quantities and galactose and lactose in the smallest; the hyperthyroid rats followed a similar pattern of preferences (largest quantities of maltose and glucose and the smallest amounts of galactose and lactose).

Figure 28–4 shows for the hyperthyroid rats the average intake at the various concentrations of the six sugars in ratios of calories per kilogram body weight of the sugars to calories per kilogram body weight of the food. Maltose had the highest ratio; then came glucose, sucrose, fructose, galactose and lactose in descending ratios.

Comparison of rankings of the various sugars on basis of intake with their rankings on basis of various characteristics shown in Table 28–I failed to establish any agreement with molecular weight, solubility, sweetness, intestinal absorption rate or hyperglycemia; but it did bring out very close agreement with the "limits of assimilability." Table 28–III shows this relationship in more detail for concentrations of the sugars from 9–30 per cent inclusively. To make possible comparison of ranking of sugars on basis of intake and their limits of assimilability, values of both are represented by line scales of the same length—the sugar with the highest value in each instance being at the top. The first scale at the left shows the daily intake of the six sugars in grams per kilogram for the 54 normal rats; the second scale shows the intake in grams per kilogram for the 21 hyperthyroid rats; the third shows ratios of sugar to food intake in calories per kilogram body weight. These ratios agree very closely with the ranking of sugars for limits of assimilability, grams per kilogram body weight, determined first by Mendel and Jones [4] on normal rabbits and then by Ariyama and Takahasi [1]

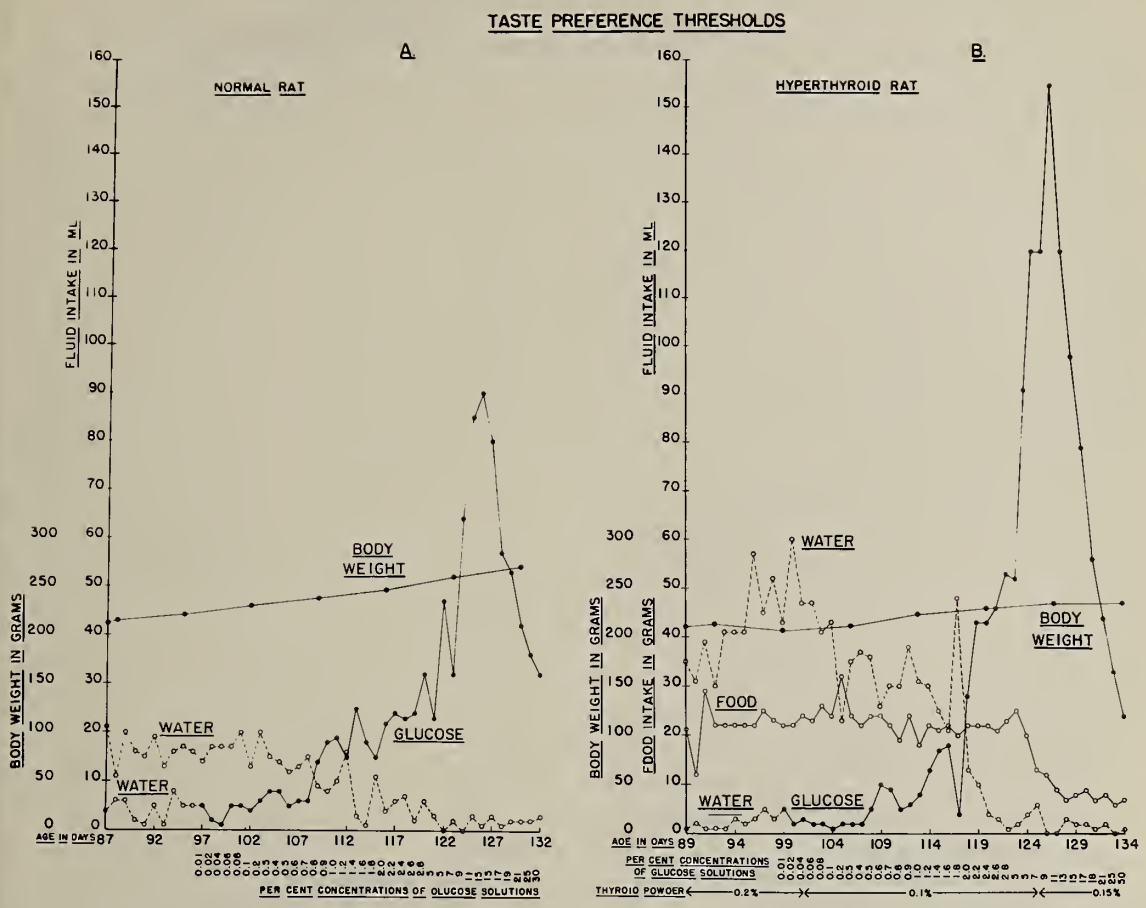


Figure 28–2 Graphs of taste preference curves for glucose for an individual normal rat (A) and an individual hyperthyoid rat (B) showing parts of curves (concentrations–9–30 per cent) used in determining maximum intake of the various sugars.

on normal rats. The ranking is so close it leaves little doubt that the amount of sugars ingested by the rats is determined by the "limits of assimilability" of the sugars.

RATS EAT FOR CALORIES

Further study of the data obtained from these taste threshold studies brings out a principle of self-selection of diets—rats eat for calories. It was found in the present experiments in which rats had a choice of sugar solutions and stock diet, that as they increased their caloric intake from increasing concentrations of the sugars, they decreased, in direct proportion, their caloric intake from the stock diet. As may be seen in Figure 28–5, their total caloric intake remained the same.

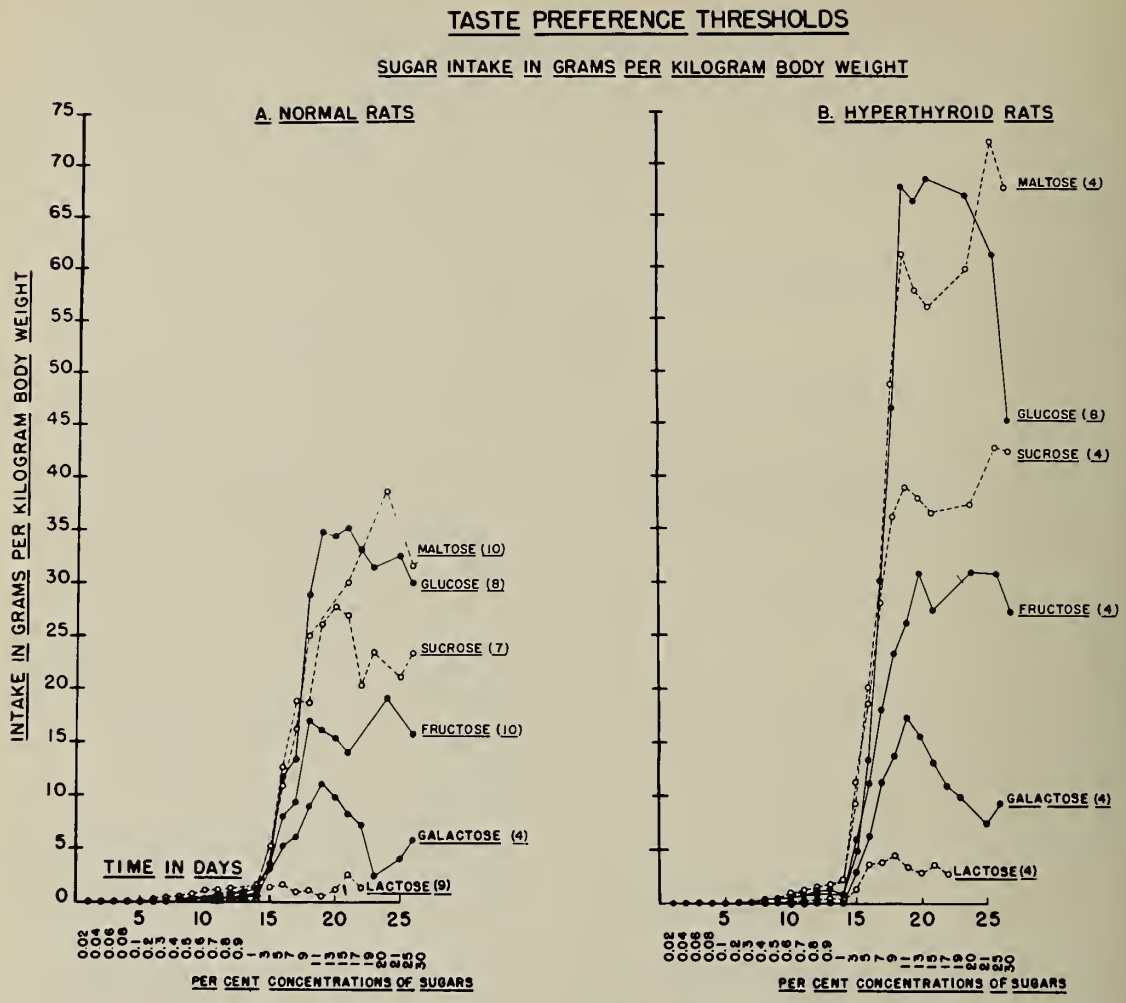


Figure 28–3 Graphs for normal rats (A) and hyperthyroid rats (B) showing an average daily intake of the six sugars at concentrations from 9–30 per cent inclusively in grams per kilogram body weight.

NEUROLOGY OF "LIMITS OF ASSIMILABILITY"

At present we have no idea about location of mechanisms in the brain that make the rat stop eating when the limit of assimilability is reached.

Knowledge of this mechanism might throw light on the problem of obesity.

NUTRITIVE VALUES OF SIX COMMON SUGARS

Results of the present experiments demonstrated the great differences in appetites shown by rats for the six common sugars.

It remains to be shown whether the appetite levels bear a direct relationship to the nutritive value of the sugars.

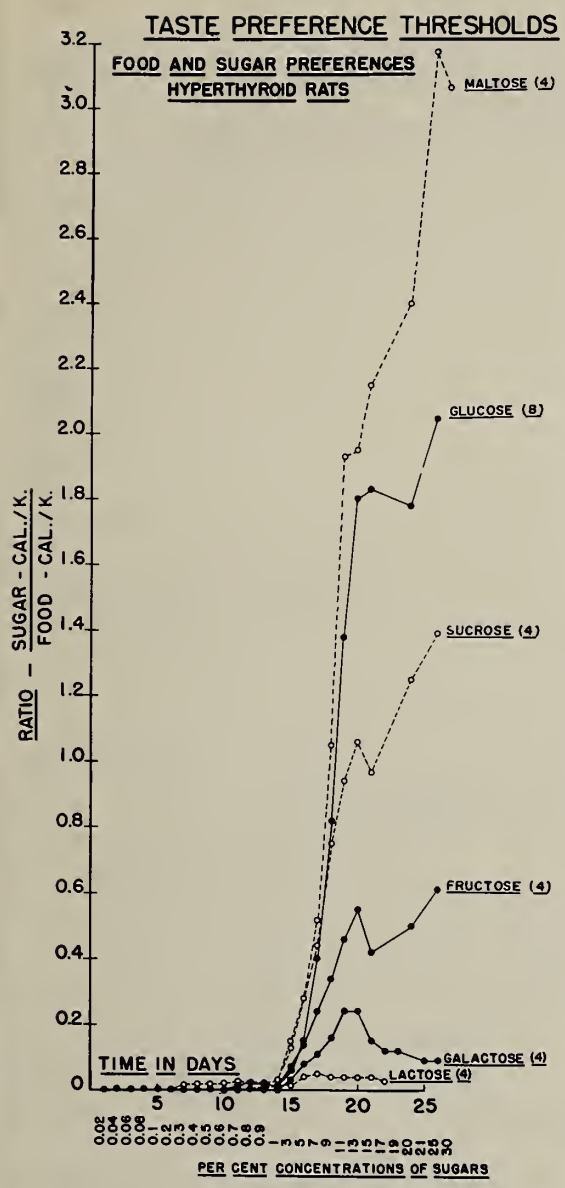


Figure 28–4 Graphs showing average intake of six sugars in concentrations of 9–30 per cent for hyperthyroid rats.

To determine the nutritive values we used what we have called the "single-food choice" method: rats of a standard age and weight were restricted to one sugar and water or one sugar and a 0.02 per cent solution of thiamine hydrochloride. Survival time was used as a measure of the nutritive value. Records were also taken on spontaneous running activity, food and fluid intake and body weight.

One example should suffice to illustrate this technique. Figure 28–6 shows the daily total activity of a normal rat kept on our stock diet and then placed on a single-food diet of dextrose. On dextrose alone this rat survived 36 days; on dextrose and the solution of thiamine hydrochloride the other rat survived 76 days. Both rats lost weight at constant rates on the single-food choice diet, but they remained very active—running as much as 10–15 miles per day for weeks. Although these rats received no protein, no fat, no minerals or vitamins except B^1 they showed no signs of deficiency—no loss of teeth or hair. Clearly dextrose by itself must have a very high energy value. Maltose, sucrose, and fructose showed similar records.[8]

How do these results square with the general belief in the harmful effects of the sugars?

Results of these and other experiments from my laboratory indicate that harmful effects produced by ingestion of sugars do not result from the sugars themselves, but from other ingredients of the diet. Thus, for example, while rats on a normal diet but lacking in magnesium will show audiogenic fits in 10–14 days and also other signs of deficiencies, none of these symptoms appeared in rats kept on the single-sugar choice diet during their lifetimes of 60–75 days.

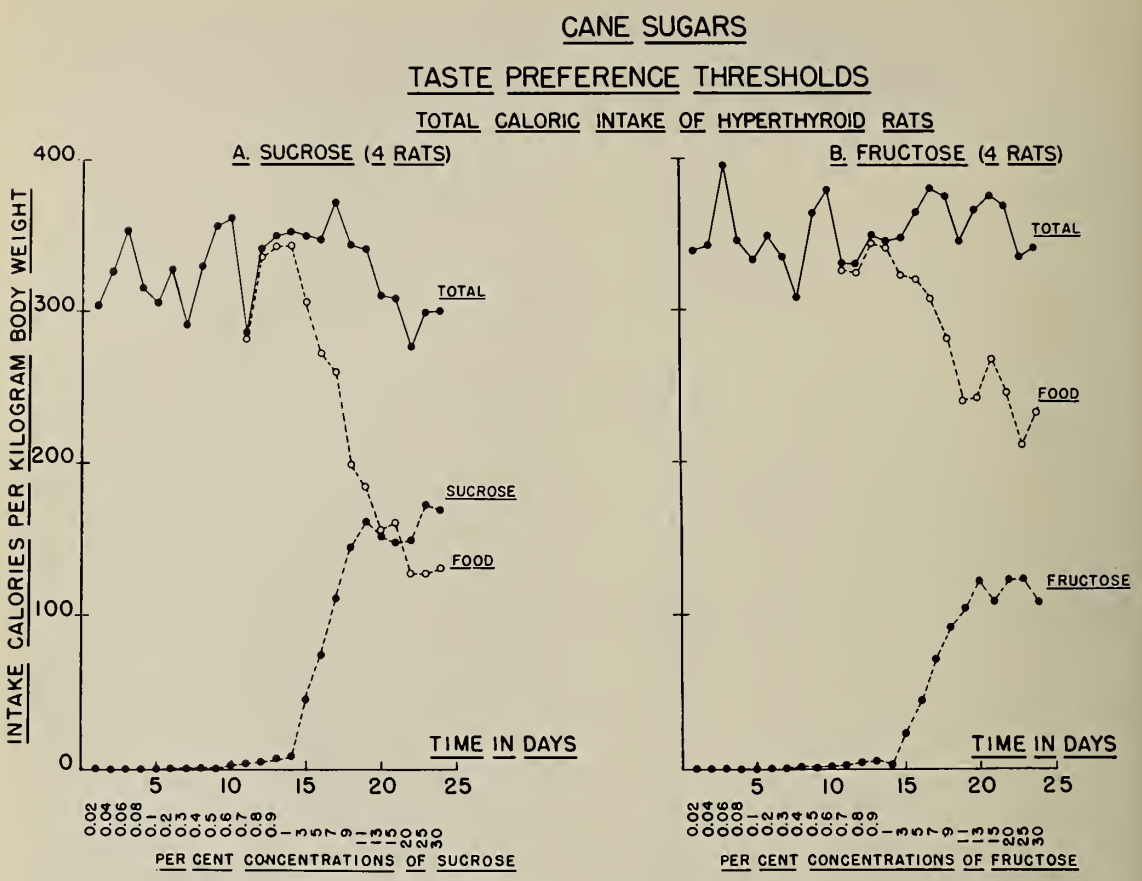


Figure 28–5 Graphs: A-showing caloric intake from sucrose and stock diet, and total caloric intake (from sucrose and food) on the successively higher concentrations of sucrose in this taste preference test. B-same for fructose.

In summary, results of our observations on the sugars have demonstrated the existence of three principles on self-selection of diets:

1. Taste may determine whether a rat will ingest a sugar.
2. "Limits of assimilability" determine how much of a sugar the animal will ingest.
3. Under a wide range of conditions rats eat for calories. This was first demonstrated in experiments on alcohol.[10] It is almost impossible to force a normal rat to eat more than its fixed limit of calories.

These results have also demonstrated the high levels of assimilability of maltose, particularly in hyperthyroid rats; and the definitely lower and consistent maltose taste thresholds in both normal and hyperthyroid rats.

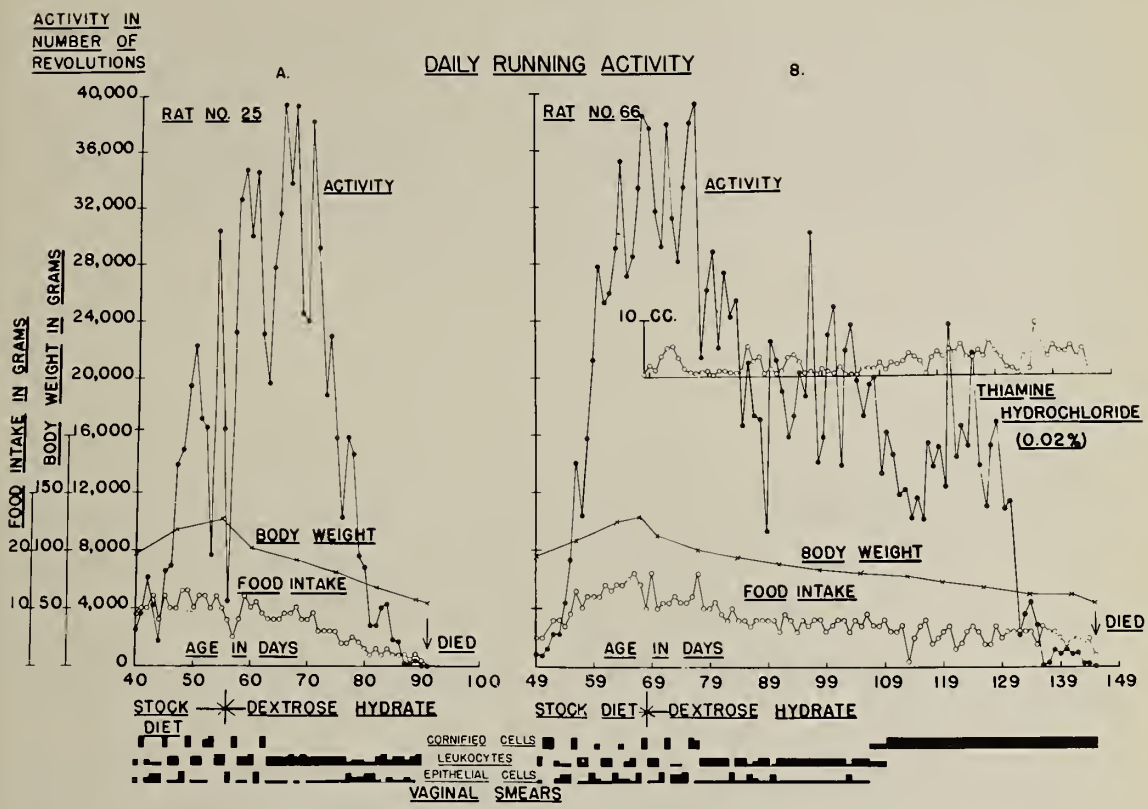


Figure 28–6. Graphs showing results of single-food experiments in which rat "A" had access only to dextrose and water; and "B" access only to dextrose and a 0.02 per cent solution of thiamine hydrochloride. Graphs show daily intake of stock diet and dextrose in grams; spontaneous running activity in revolutions of a drum; and weekly body weight. The graphs also show cornified cells, leucocytes and epithelial cells in vaginal smears. The rat on dextrose lived 36 days; the rat on dextrose and B_1 lived 76 days.

REFERENCES

1. Ariyama, H., and Takahasi, K.: Uber den relativen Nahrwert der Kohlenhydrate und verwandten Substanzen. *Biochem. Ztschr.* 216: 269, 1929.
2. Biester, A., Wood, M.W., and Wahlin, C.S.: Carbohydrate studies. I. The relative sweetness of pure sugars. *Amer. J. Physiol.* 73: 387, 1925.
3. Cori, C.F.: The fate of sugar in the animal body. I. The rate of absorption of hexoses and pentoses from the intestinal tract. *J. Biol. Chem.* 66: 691, 1925.
4. Mendel, L.B., and Jones, M.R.: Studies on carbohydrate metabolism in rabbits. I. Observations on the limits of assimilability of various carbohydrates. *J. Biol. Chem.* 43: 491, 1920.
5. Reinhold, J.G., and Karr, W.G.: Carbohydrate utilization. II. Rate of disappearance of various carbohydrates from blood. *J. Biol. Chem.* 72: 345, 1927.
6. Richter, C.P.: Salt taste thresholds of normal and adrenalectomized rats. *Endocrinol.* 24: 367–371, 1939.
7. Richter, C.P., and Campbell, K.H.: Taste thresholds and taste preferences of rats for five common sugars. *J. Nutrit.* 20: 31–46, 1940.

8. Richter, C.P., and Rice, K.K.: The effect of thiamine hydrochloride on the energy value of dextrose studied in rats by the single food choice method. *Amer. J. Physiol.* 137: 573–581, 1942.
9. Richter, C.P.: Self-regulatory functions during gestation and lactation. Gestation-Transactions of the Second Conf., March 8, 9, 10, 1955, Princeton, New Jersey. Pages 11–93.
10. Richter, C.P.: A study of the effect of moderate doses of alcohol on growth and behavior of the rat. *J. exper. Zool.* 44: 397–418, 1926.

Chapter 29

ROLE OF HEDONIC PROCESSES IN THE DEVELOPMENT OF SWEET TASTE PREFERENCES

Paul Thomas Young

I will first consider some fundamental concepts and definitions. And then, describe experimental methods and typical results relating to the role of hedonic processes in development of sweet taste preferences. And, finally, some theoretical discussion.

CONCEPTS AND DEFINITIONS

Appetitive, neutral and aversive effects of stimulation

Sensory stimulations elicit positive, neutral, and negative effects. Positive effects are *appetitive* leading the organism to continue and repeat the conditions that produce them. *Neutral,* or nearly neutral, hedonic effects are found in observant, exploratory, and manipulative behavior. Neutral effects occur also in some reflexive, habitual, automatic reactions. Negative effects are *aversive* leading the organism to terminate, escape from, and avoid the conditions that elicit them.

These appetitive, neutral, and aversive effects depend upon neural structures that are the final product of millions of years of organic evolution. There are built-in neural mechanisms that regulate behavior. For example, many organisms have innate neural structures that make a sweet taste appetitive, pleasant, and a bitter taste aversive, unpleasant.

To represent adequately the positive and negative effects of stimulation, and the pleasantness and unpleasantness which we feel, two dimensions are needed. (See Figure 29–1.) One dimension is the level of activation—the degree of excitement produced by a stimulating situation. The other dimension represents the hedonic continuum. This is a bipolar continuum with positive affective arousals at one extreme and negative at the other. Arrows in Figure 29–1 represent two directions of hedonic change: toward the positive pole or toward the negative. Affective processes run a temporal course. They vary in sign (positive or negative), intensity, and duration.

Sensory and preferential discrimination

A distinction can be drawn between *sensory* and *preferential* discrimination. We can easily discriminate between black and white or between sweet and

bitter tastes. These discriminations are sensory, not preferential. Preference can be defined as an *evaluative* choice.

In studies of food preferences animals are offered a choice. The selection of one food rather than another may depend upon its position or upon a habit of choice that has already been set up. In many cases, however, the choice depends upon the relative palatability of the test foods. Palatability can be defined as the hedonic response to a food.

The hedonic response may be pleasant, appetitive or unpleasant, aversive. There are varying degrees of positive and negative palatability.

It is my thesis that the immediate hedonic effect of sensory stimulation organizes behavior and leads to the development of preferential behavior. With human beings the pleasantness or unpleasantness of contact with foodstuffs determines acceptance, rejection, and preference. With non-human animals palatability also determines choice and palatability can be demonstrated within a strictly objective frame of reference.

There are also delayed hedonic effects that can influence choice. Subjective feelings of comfort and discomfort follow the ingestion of certain foods. We are familiar with the "green apple" effect. With non-human animals there are deferred beneficial or detrimental effects that, through memory, can become associated with the ingestion of particular foods. If an animal associates ingestion of a particular food with some beneficial effect or with relief from a detrimental effect, a habit of dietary selection may become established. If the deferred hedonic effect is negative, as with toxic substances, aversive behavior may develop. Hedonic effects presumably regulate behavior at the intraorganic level as well as the oropharyngeal level.

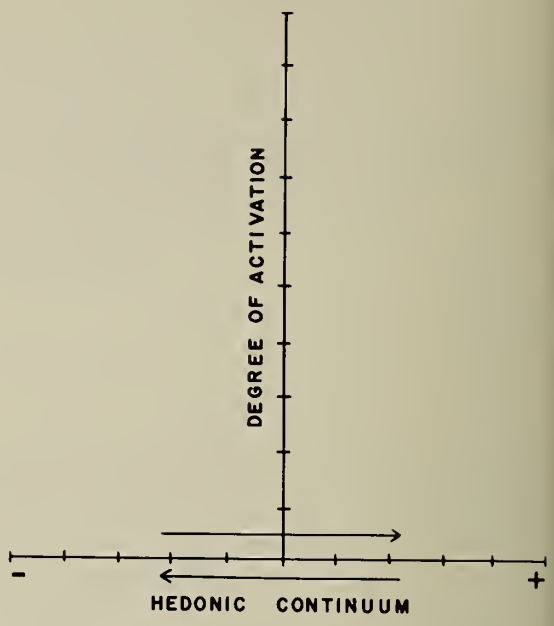


Figure 29–1. Two dimensions of motivation: activation and hedonic value.

Therefore, the choice of foods may be determined by immediate or deferred hedonic effects or by established habits. With man the choice of foods may also rest upon rational grounds. A person decides that one course of action is more desirable than another. Rational considerations influence choice and preference. If people are told that dental caries follow the ingestion of sugar or that smoking is injurious to health, the information will to some extent influence behavior. But rational decision must contend with powerful instinctive tendencies, with addictive habits, and acquired tolerances. Sugar is innately sweet and pleasing. This is a fact of life.

EXPERIMENTAL METHODS AND RESULTS

The brief-exposure preference tester

The brief-exposure preference tester was used extensively in our early studies of the food preferences of rats. Figure 29–2 is a floor plan of the apparatus. Two test foods (F), solid or liquid, are presented in tubes or cups at the center of an open field. A rat is placed in the starting box (B) with the door (D) closed. After a brief delay in the box the door is raised and the rat is free to approach the test foods. The behavior of the animal is observed through a window (W).

When a rat has accepted one and the same food from 1 to 4 seconds both foods are lowered out of reach and their positions interchanged for the next run. A preference is recorded for the food that first elicited sustained contact.

The rats are trained to shuttle back and forth between box and foods. Being rewarded by a sip or nibble of the incentives on each round trip, the animals soon learn the shuttle pattern. During a session of ten to fifteen minutes it is usually possible to observe a significant series of choices. Some of the series reveal position habits—the animal repeatedly taking the food at his right or left. Other series clearly show the development of consistent food preferences.

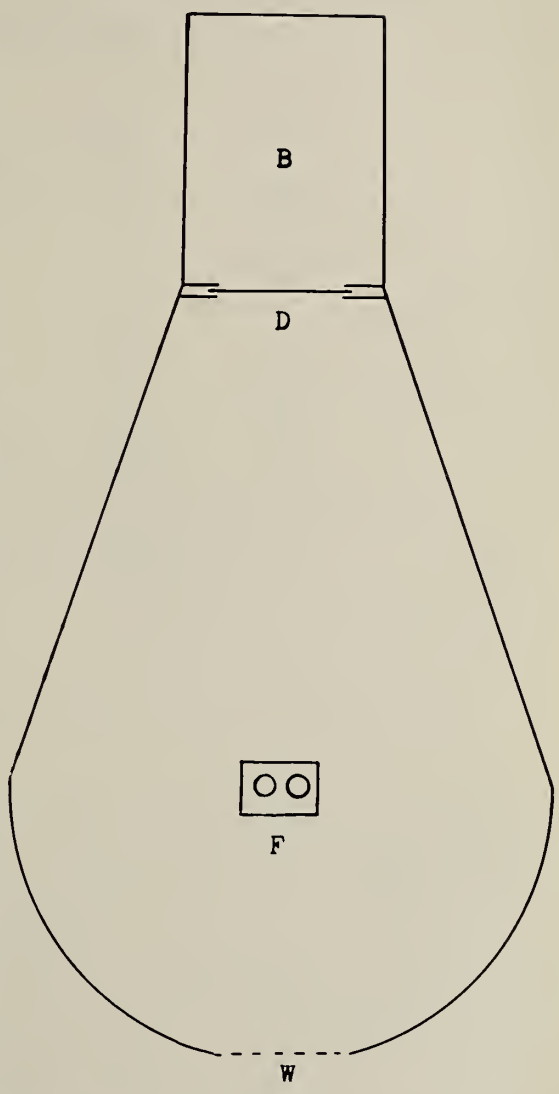


Figure 29–2. Floor plan of the brief-exposure preference tester.

Now consider some of the findings in early studies with the brief-exposure preference tester:

Preferential hierarchies. Preference tests with solid and liquid foods have repeatedly shown that test foods arrange themselves into a hierarchy from low to high levels of acceptability. The series is transitive. If food A is preferred to B, and B to C, then A is preferred to C. When exceptions to this rule of transitivity occur, we are safe in assuming that the conditions that determine acceptability have changed.

Figure 29–3 shows the preferential hierarchies of ten rats tested with all possible paired comparisons of six

Preferential Hierarchies of Ten Rats Tested with Six Foods

Rat	Preferential Hierachy											
40	S	>	M	>	W	>	D	>	B	>	F
41	M	>	S	>	W	>	D	>	B	>	F
42	M	>	S	>	D	>	W	>	F	>	B
43	M	>	S	>	D	>	W	>	F	>	B
44	M	>	S	>	W	>	D	>	B	>	F
45	M	>	S	>	W	>	D	>	F	>	-*
46	M	>	S	>	W	>	D	>	F	>	B
47	M	>	S	>	W	>	D	>	B	>	F
48	M	>	S	>	W	>	D	>	F	>	B
49	M	>	S	>	W	>	D	>	F	>	B
Group	M	>	S	>	W	>	D	>	F	>	B

*Died before B vs F test.

Figure 29–3. Preferential hierarchies of ten rats tested with six foods. *From* Young (1933).

foods.[12] The foods are symbolized by letter as follows: M = fresh milk, standardized at 4% butterfat; S = granulated cane sugar, extra fine; W = whole wheat powder, freshly ground; D = dehydrated milk (a commercial product known as KLIM—which is milk spelled backwards); F = commercial white flour made from wheat; and B = butterfat prepared for the experiment. The striking feature of this chart is the uniformity of preference within the group of subjects. Individual differences in preference do occur but they are always between two foods that are close together in the hierarchy for the group as a whole.

Preferential trends and shifts. In an early study with the brief-exposure technique, sugar was used as a standard which was tested against four other foods.[12] The comparison foods, in order, were flour, wheat, butterfat, and milk.

Figure 29–4 shows the percent of choices made for each pair of foods by a group of three male rats. Percentages greater than 50% indicate the preferred food. Inspection of this figure reveals several outstanding features: (1) In every series of tests there is a consistent preference. Sugar is preferred to flour, to wheat, to butterfat. Milk is preferred to sugar. (2) In every series of successive tests there is a preferential trend, i. e., a more or less steady increase in the percent choices of the preferred food. The trend shows the gradual development of a preferential habit. The slope of the trend, as we shall see, depends

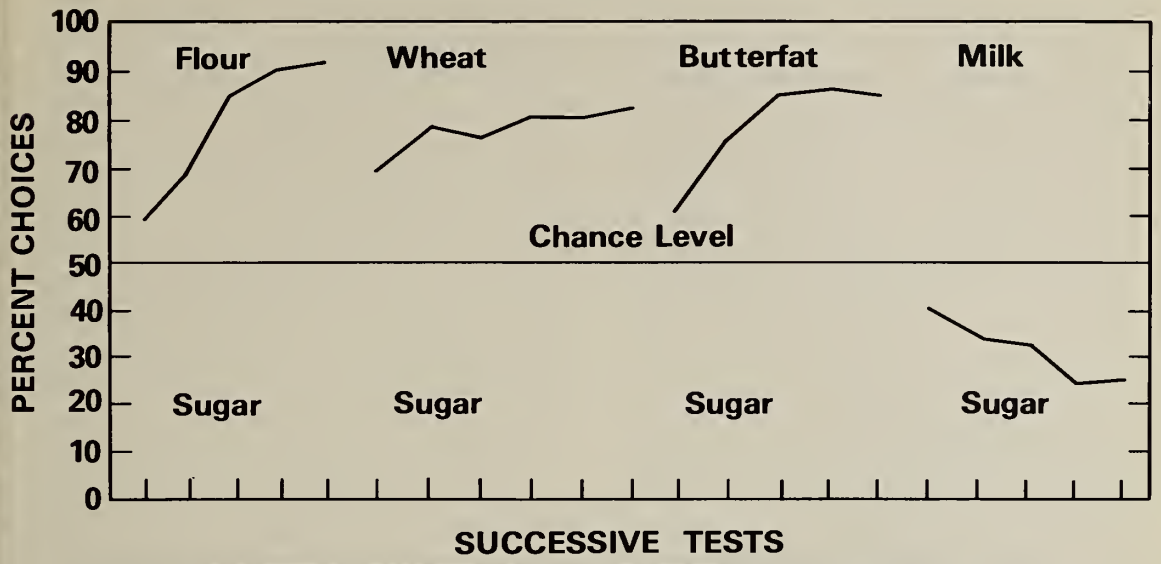


Figure 29–4. Percent choices of sugar (sucrose) tested against flour, wheat, butterfat and milk during successive tests. *From* Young (1933).

upon the *difference* in palatability between the pair of test foods. (3) When one test food was suddenly substituted for another there was an abrupt shift in the percent of choices. These abrupt changes, I believe, are motivational. They reflect a dynamic difference of arousal between two incentives. (4) The more gradual preferential trends indicate learning. They show the cumulative effects of repeated trials with differential reinforcements.

In a later study preference tests were made with three foods of known palatability: cane sugar (S), wheat powder (W), and casein (C).[14] (See Figure 29–5) Sugar is preferred to wheat. Both foods are highly acceptable and the difference in palatability between them is small. Casein is accepted by rats but the palatability level is low. Hence the preferential hierarchy is: S > W > C.

In this study every possible pair of foods was tested for preference.

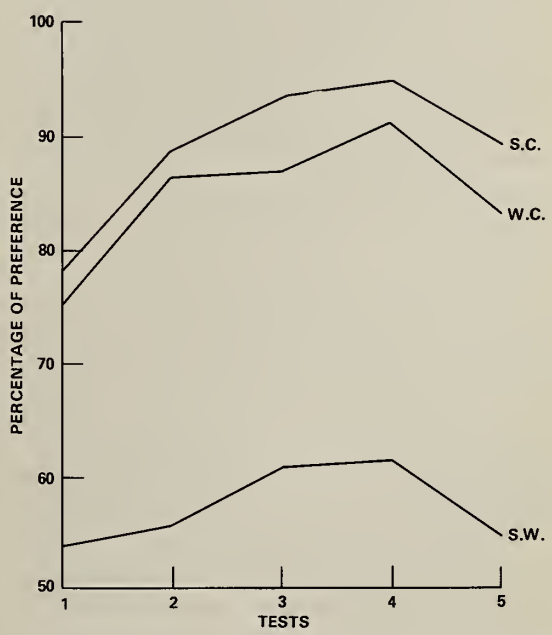


Figure 29–5. Stability of the hierarchy when practice in preferential discrimination is equally distributed with pairs of test foods. Sugar (S) is slightly more palatable than wheat (W). Both S and W are much more palatable than casein (C). *From* Young (1947).

Cycles of preference testing were arranged so that no food had any advantage of position or priority. There were five successive cycles of preference tests. During the last cycle an epizootic of respiratory disease swept through the colony of 30 rats but preference testing was continued.

Figure 29–5 shows the results of the experiment. The preferential trend for sugar vs. wheat is not far above the 50% chance level. Sugar is preferred to wheat but the difference in palatability between these foods is relatively small. The curves for sugar vs. casein and wheat vs. casein are relatively high in percentage of preference. Both sugar and wheat are very much more palatable to rats than casein.

Now the outstanding feature of this chart is that the preferential hierarchy remains unchanged when practice is equally distributed among the three pairs of test foods. Even the epizootic during cycle 5 did not change the preferential hierarchy. When sick, the rats were slow in running and less discriminating between pairs of test foods but the preferential hierarchy was stable.

A general conclusion from this study is that the preferential trend depends upon repetition or practice in making a discrimination. This is a process of learning. The hedonic relations, however, are independent of learning. They are relatively stable.

Psychophysical studies with solutions of sucrose and saccharin

One of the simplest and best methods of testing the preferences of rats was employed by Dr. C. H. Madsen.[21] In this method the animal is confined to a small cage with two stainless steel nozzles at the front. The nozzles are connected to burettes that contain the test fluids. During an actual test, for example, one burette might contain a sucrose solution of 1% and the other a solution of sodium saccharin of 0.4%.

Figure 29–6 shows one unit of an electronic preference tester that records separate tongue contracts cumulatively but Dr. Madsen did not use the electronic device. Instead he used a direct manual method of presentation and timing:

At the start of a trial, one nozzle is inserted into the cage. After the rat has taken a few licks this nozzle is withdrawn and the other inserted for a few licks. Immediately after this forced sampling both nozzles are inserted together. The rat is now free to

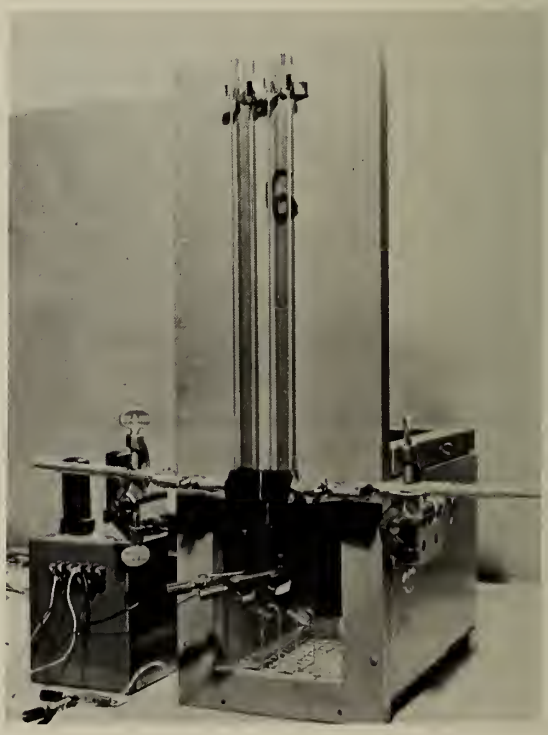


Figure 29–6. One unit of an electronic preference tester.

sample the fluids back and forth or to accept one rather than the other. Observation continues until one and the same fluid has been accepted continuously for 5 to 10 seconds. Then both nozzles are withdrawn and a preference recorded for the fluid that first elicited sustained drinking. Timing is controlled by a series of auditory signals at 5–second intervals.

In a series of trials the relative positions of burettes and the order of preliminary sampling are staggered to control space and time factors.

To determine hedonic equality of two fluids the psychophysical up-and-down method is employed.[3] With this method the sucrose concentration is varied up and down from trial to trial. From the data the sucrose concentration that yields hedonic equivalence can be computed.

Hedonic equivalence of solutions of sucrose and sodium saccharin. Figure 29–7 shows the hedonic equivalence function for solutions of sucrose and sodium saccharin. Vertical lines crossing the curve indicate the range of individual measures of hedonic equality for a group of eight rats tested individually. The solid curve is the mean hedonic equivalence function for the group as a whole. On the basis of this curve I estimated that the optimal concentration for solutions of sodium saccharin is about 0.5%. And this optimal concentration is hedonically equivalent to a sucrose solution with a concentration of 3.5%. Incidentially, human observers report that sodium-saccharin solutions are sweet and pleasant up to a concentration of about 0.5%; higher concentrations become sharp and unpleasant.

Isohedons in the sucrose-saccharin stimulus area. A *stimulus area* represents an infinite number of compound solutions containing two solutes. In Figure 29–8, for example, the stimulus area is defined by the concentrations of sucrose and sodium saccharin. An *isohedon* is a curve showing the location within the area, of compound stimuli that are hedonically equal.

To obtain data for plotting an isohedon we make a series of preference tests. In every trial there is a standard sucrose solution with a constant concentration and a comparison solution with a fixed concentration of saccharin. The sucrose concentration in the *compound* is varied up and down from trial to trial. From the data it is possible to compute the sucrose concentration in the compound that makes the compound isohedonic with the simple sucrose standard.

Figure 29–8 shows a family of isohedons with sucrose standards of 4, 8, 16, and 32 percent (4, 8, 16, 32 CHO). Each isohedon makes a dip where the saccharin concentration in the compound is 0.5%. The dip indicates that the presence of saccharin in the compound makes a positive contribution to palatability. Concentrations above this optimal value increasingly impair the palatability of the compound.

The location of optimal palatability in this family of four isohedons agrees with the location in the hedonic equivalence function.

Isohedons have been plotted in several other gustatory stimulus areas and in an auditory area of pure tones defined by the frequency and energy of the auditory stimulus.[4,17] In general, these psychophysical studies show: (1) That positive and negative

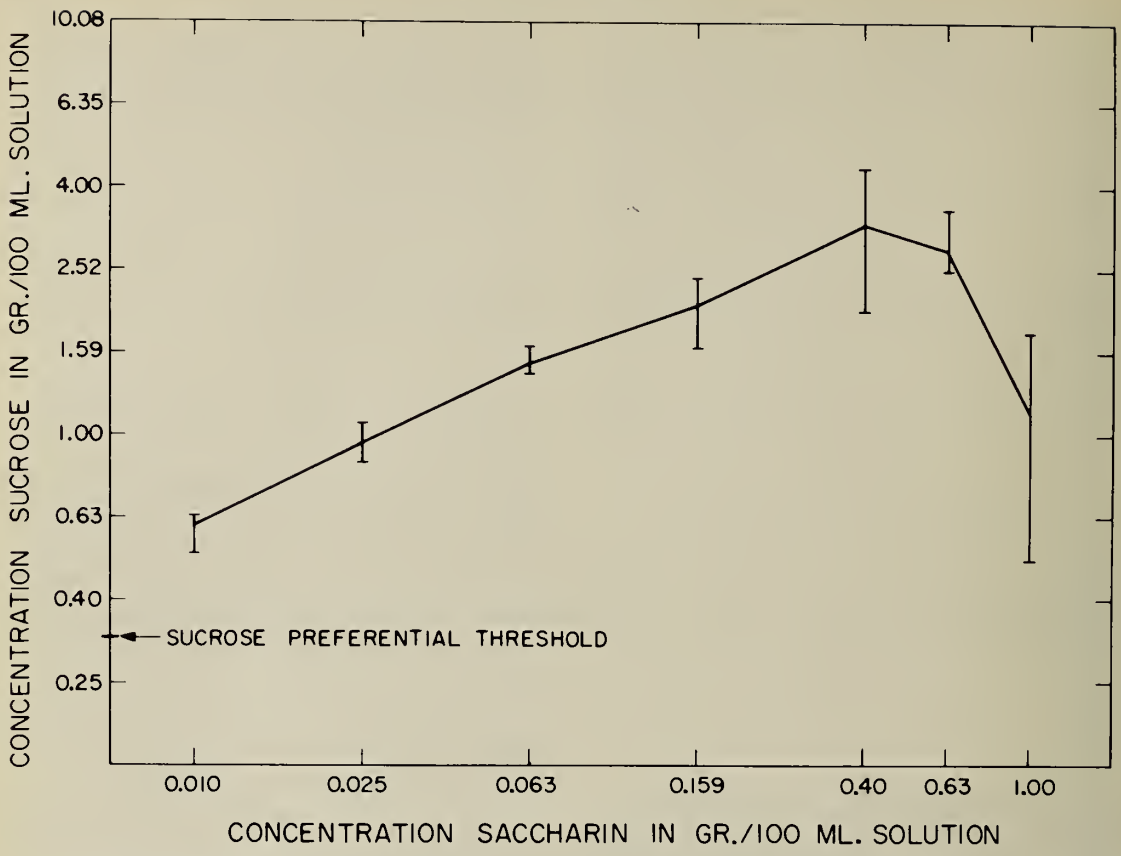


Figure 29–7. Hedonic equivalence of solutions of sucrose and sodium saccharin. *From* Young and Madsen (1963).

affective arousals summate algebraically. (2) That hedonic integration occurs across sensory modalities. For example, a shock on the foot delivered simultaneous with gustatory contact has the same influence on palatability as the addition of quinine to the taste solution. (3) That hedonic integration follows precise laws of combination that are independent of sensory laws.

Let us now consider another method of testing preferences:

The intake preference method

The intake method of studying preferences has been used extensively by Richter and many others. The method will be illustrated by two figures borrowed from Richter.[11]

Figure 29–9 shows several individual cages. Each cage is equipped with two or more graduated bottles containing test fluids. The rat is offered a continuous choice of the fluids. The experimenter takes a reading of the volume of fluid in the tubes before exposure and again, 24 hours later, after exposure. From the two readings the daily intake of a fluid can be determined.

A typical result is shown in Figure 29–10. The test fluids were two sugars: sucrose and maltose. At the start of the experiment both graduated bottles were filled with distilled water and the positions were

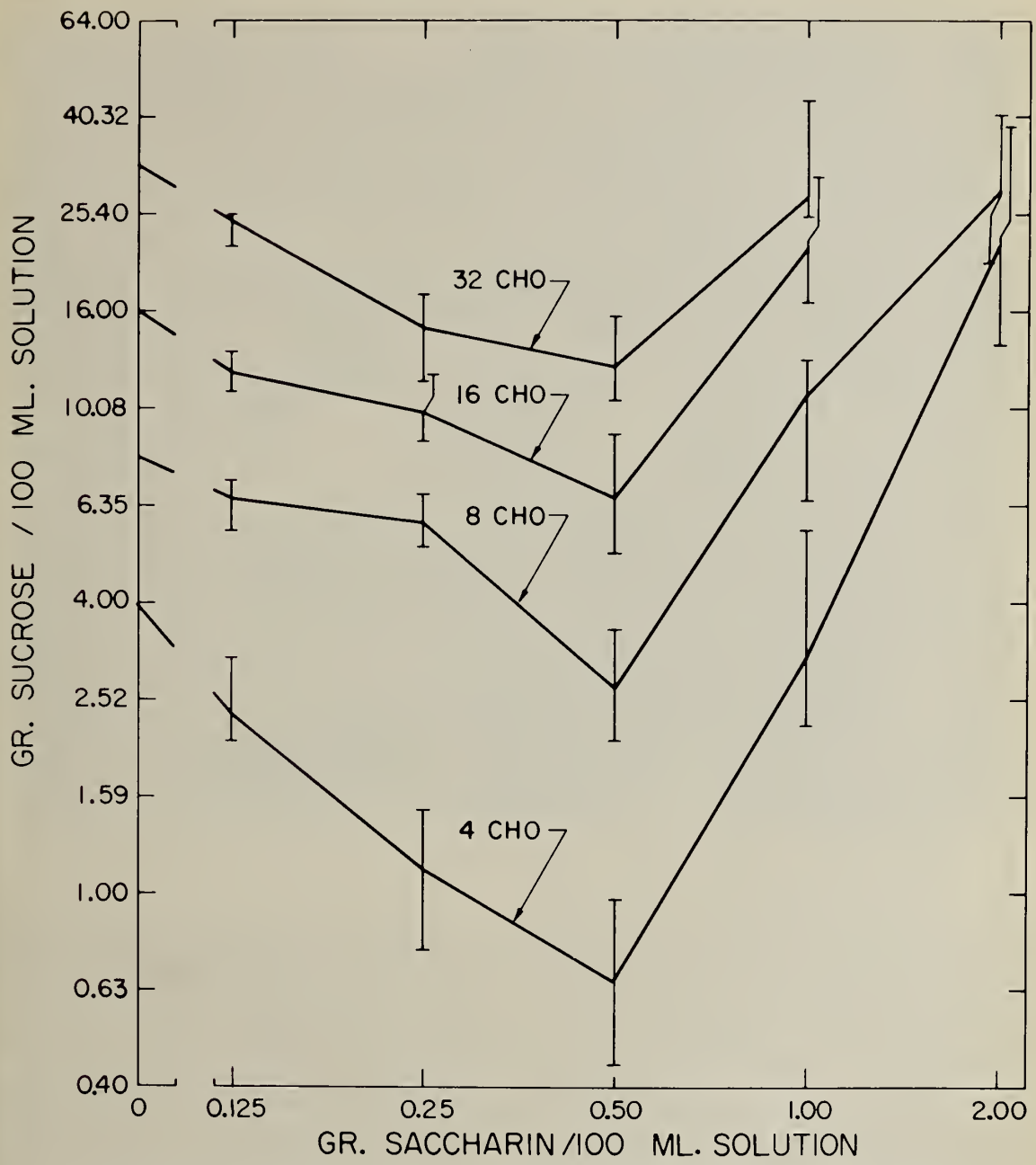


Figure 29–8. Isohedons in the sucrose-sodium saccharin stimulus area. Sucrose (CHO) hedonic standards were 4, 8, 16, 32 percent. *From* Young and Madsen (1963).

interchanged from day to day. Then a subliminal concentration of sugar solution was presented in one of the bottles. The sugar concentration was gradually increased, day by day, throughout the experiment and daily measures of intake were made.

The illustration shows the average daily fluid intake for a group of rats. There are three critical concentra-

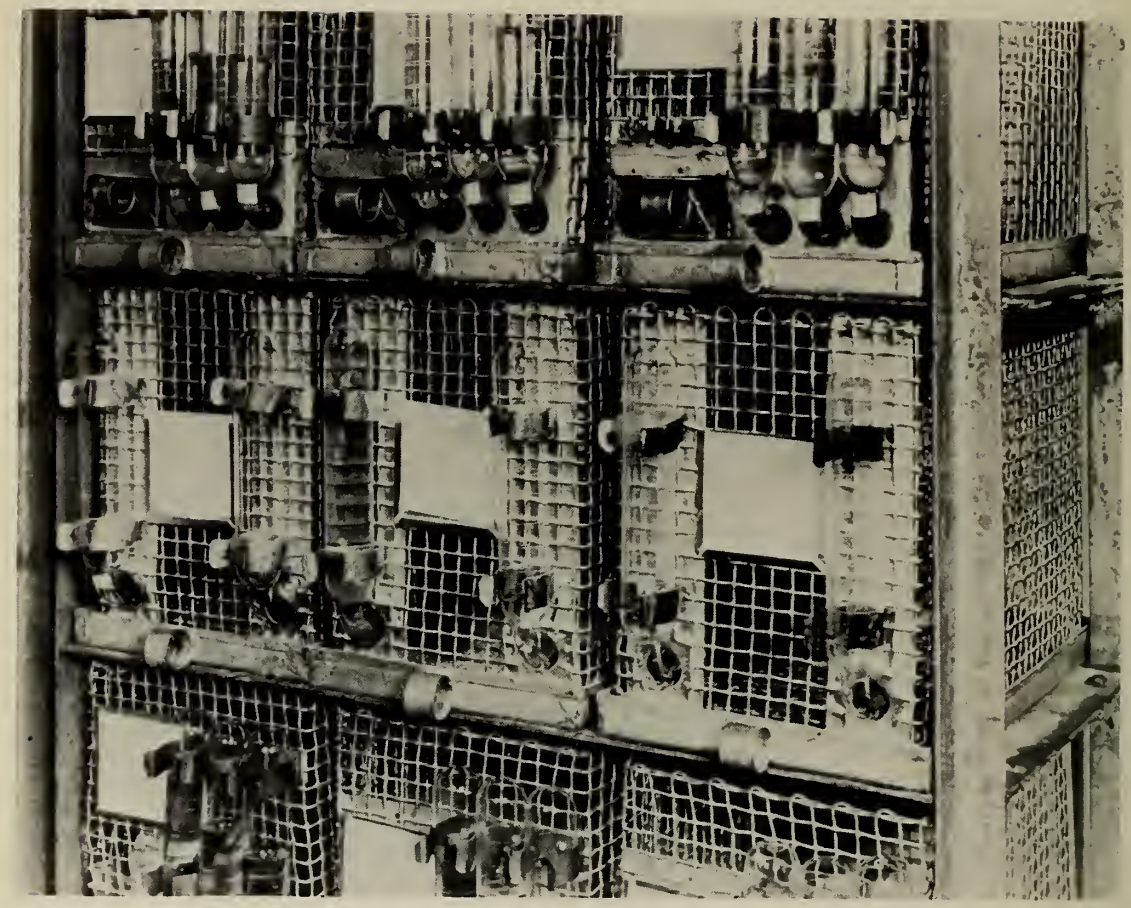


Figure 29–9. Individual cages for testing preference with the continuous intake method. *Courtesy of* Dr. Curt P. Richter.

tions. First, the preferential threshold is the concentration at which the animals begin to show a significant preference for the sugar solution. Second, there is the concentration (about 10% for sucrose) at which intake of the sugar solution is maximal. Third, there is the indifference concentration—a concentration above the threshold at which the intake of sugar solution and distilled water are again equal.

Some pairs of fluids, especially when one fluid is hedonically negative, do not show a peak of maximal intake and an indifference concentration. Many toxic substances are consistently rejected at threshold.

Richter has stated that: "*Solutions preferred in any concentration to water by the rats are apt to have nutritional value, while those which are not preferred to water in any concentration are apt to have no nutritional value or to be toxic.*"

Further, Richter has claimed that if rats are maintained on the components of a complete and fully adequate diet, presented in separate containers, they are able to select and balance a diet that yields normal growth, reproduction and health.[9,10] The animals are equipped with self-

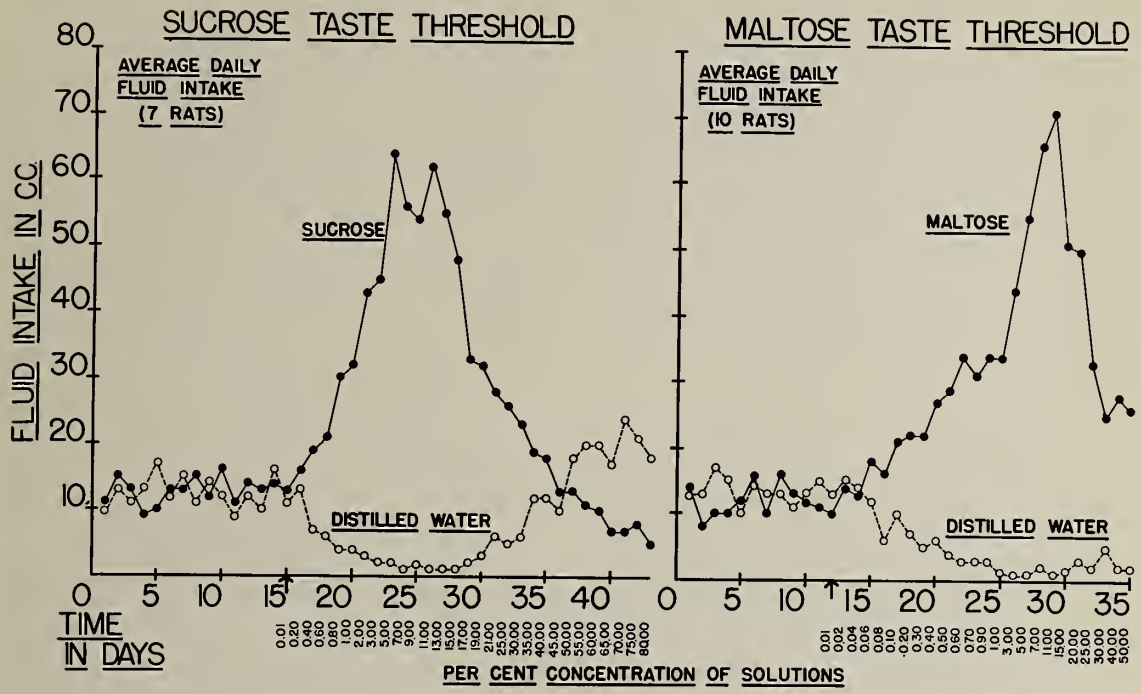


Figure 29–10. Average daily intake of sucrose and maltose tested with the continuous intake method. *Courtesy of* Dr. Curt P. Richter.

regulatory mechanisms that enable them to meet metabolic, homeostatic, and nutritional needs. This doctrine that a self-selected diet is nutritionally adequate, however, has been the subject of considerable controversy.

Nutritional need, habit and preference. To study the relation between nutritional need and preference Dr. J.P. Chaplin and I maintained groups of rats in large cafeteria cages upon an eleven-component self-selected Richter diet.[20] Every animal in the group had unlimited access to the eleven components which were presented in separate containers. Two of the components were sucrose—a source of carbohydrate and energy—and casein—a source of protein which is necessary for normal metabolism and growth.

From birth the rats were maintained on a stock laboratory diet. When placed in the cafeteria on the first day they ingested excessive quantities of sucrose and casein and, incidentally, they developed diarrhea. After a few days for adjustment to the cafeteria diet the average daily intake of sucrose and casein stabilized. The daily intake of casein was consistently higher than that of sucrose as shown by 10-day averages. If relative intake measures preference, we would have to conclude that casein is preferred to sucrose.

At the start of the experiment all rats were given a brief-exposure preferences test. There was a uniform preference of sucrose to casein. Preference tests were repeated at different stages of the experiment. The result was always the same: sucrose is preferred to casein.

A metabolic and nutritional need for protein was then created by re-

moval of casein from the cafeteria maintenance cages. Protein starvation steadily increased day by day. Despite a growing need for protein the animals developed a preference for sucrose when tested with the brief-exposure technique. After 29 days of protein starvation the need for protein was severe and obvious but the rats increasingly developed a consistent preference for the sweet-tasting sugar.

At this stage of the experiment we tested the rats individually in the cafeteria cage. The hoppers containing sucrose and casein were widely separated and in familiar locations. Each animal made a series of runs to sucrose or to casein, being rewarded by a brief nibble of the food selected. The test foods were far apart and the rat had to choose between a run to casein or a run to sucrose.

Under these conditions all animals promptly and consistently developed a running-to-casein preference. For a while we could demonstrate two simultaneous and incompatible choices! When placed on the brief-exposure apparatus with foods close together and positions altered from trial to trial the rats preferred sucrose. A few minutes later when tested with foods in fixed familiar positions the rats preferred casein. Now simultaneous incompatible "preferences" require an explanation!

We assumed that there are two kinds of preference: one based on palatability and the other based on organic need and appetite. We assumed that our result was a function of the method of testing preferences.

Then followed a tedious series of control experiments in which we compared the method where foods are close together and positions changed from trial to trial with the method with foods apart in fixed positions as in the T maze.[13] The net result was negative. Under identical conditions of deprivation and practice the foods-together and foods-apart methods yielded the same preferences. Consequently we assumed that a *habit* of preferring sugar had developed on the preference tester and that this habit persisted during protein starvation. The true preference for casein developed on the other apparatus in complete agreement with metabolic and nutritional needs.

I formulated this principle: *Dietary habits tend to form so that they meet metabolic and nutritional needs but established habits persist even though opposed to bodily needs.* The function of dietary habits is to stabilize intake.

This conclusion was verified by Mr. Tatsuhiko Tomita in Tokyo (personal communication) who repeated the experiment under slightly changed conditions. (See Figure 29–11.) Mr. Tomita used the brief-exposure technique for tests with casein and two sugars—sucrose and glucose. He found that sucrose was preferred to glucose and both of these sugars were preferred to casein. He then introduced a period of casein deprivation. At first the preferential hierarchy did not change. Then the acceptability of casein rose first above glucose and then above sucrose. When casein deprivation was terminated the preferential hierarchy did not immediately change. Later, however, the acceptability level of casein declined first below sucrose and then below glucose to its original position.

The view that homeostatic needs influence palatability agrees, of

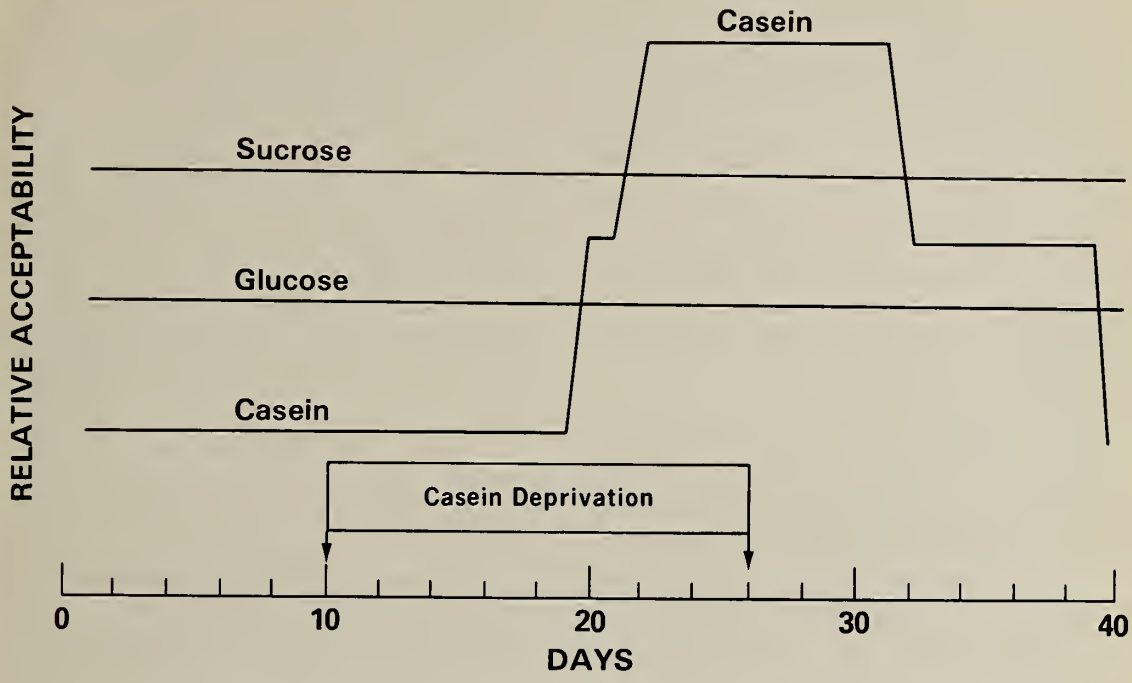


Figure 29–11. Change in the relative acceptability of casein produced by casein (protein) deprivation. Redrawn after Mr. Tatsuhiko Tomita.

course, with common sense. We know that a sip of water "tastes better" when thirsty than when satiated by drinking and that a beef steak "tastes better" when hungry than after a banquet.

The stabilizing role of food habits

Consider now the role of food habits in stabilizing intake.

During the early days of vitamin research L.J. Harris and co-workers, in England, performed some interesting experiments on the self-selection of diets by laboratory rats.[5] These workers maintained rats on a basal diet which could be made inadequate by withdrawal of the vitamin-B complex. The rats, suffering from avitaminosis, were offered a free choice among six or more supplementary foods only one of which contained a small but sufficient supply of the needed vitamin. The supplementary foods were differently flavored to aid in identification.

Intake records showed that the rats were unable to select the food containing the needed vitamin. Avitaminosis developed.

At this stage of the experiment the rats were "educated." Their "education" consisted of offering them the vitamin-containing food alone for two or three days, without choice, until they could experience the curative and beneficial effects of the diet and associate these effects with the characteristic flavor of a food. During the period of "education" the animals regained lost weight and appetite.

When the "educated" rats were again offered the original choice they continued to select the food containing the vitamin. The vitamin was then removed from this food and placed in

a differently flavored food. The rats continued to select the food that previously had contained the vitamin. Avitaminosis again developed. It was possible, however, to "re-educate" the rats by letting them experience the curative effects of a differently flavored diet that contained the needed vitamin. When given a choice they continued to select the vitamin-containing food.

These early experiments are exceedingly important for two reasons. First, they demonstrate that food habits stabilize intake quite apart from the nutritional adequacy of the food. Second, they imply that there is an hedonic basis for the organization and development of dietary habits. Relief from the distress of avitaminosis is an hedonic change away from negative affectivity and toward the positive pole of the hedonic continuum.

Reinforcement without choice

The reinforcing influence of hedonic processes is well shown in the following experiment on reinforcement without choice.[16]

To explore the relation between reinforcement and affective arousal Dr. C.L. Trafton made some tests with thirty-two naive rats in four equivalent groups of eight animals each. Each rat was given an opportunity to ingest a sugar solution for sixty seconds beginning with the first contact. Tongue contracts were counted electrically.

Four sucrose solutions were used with concentrations of 1%, 4%, 8%, and 32%. These concentrations were chosen because it had been shown that rats prefer the higher to the lower sucrose concentration when given a choice. Hedonic intensity is a direct logarithmic function of sucrose concentration.

The mean number of licks during successive 60-second tests is shown in Figure 29–12. There were eleven daily tests during acquisition followed by five tests with distilled water during extinction. During the first five days (the first 300 licks per rat) the rate of licking was proportional to the sucrose concentration. There were only two minor inversions of this relationship on day 3. The curves show that the *initial* rate of licking is proportional to the hedonic intensity (concentration) of the stimulus. After five days, however, the learning curve for the 1% group remained consistently low while the curves for the other groups cross and re-cross without significant differences presumably as these rats approached a physiological limit of licking rate.

During extinction all curves showed a drop. The drop was least for the 1% group and greatest for the 32% group. In general, the final rates of licking were inversely proportional to the initial rates. The extinction curves suggest that during acquisition the animals acquired an *expectancy* for an hedonic effect of given intensity. The small change with the 1% group is not far above a threshold value for reinforcement.

THEORETICAL DISCUSSION

At the start of this chapter I argued that to describe the appetitive and aversive effects of stimulation it is necessary to postulate two dimensions

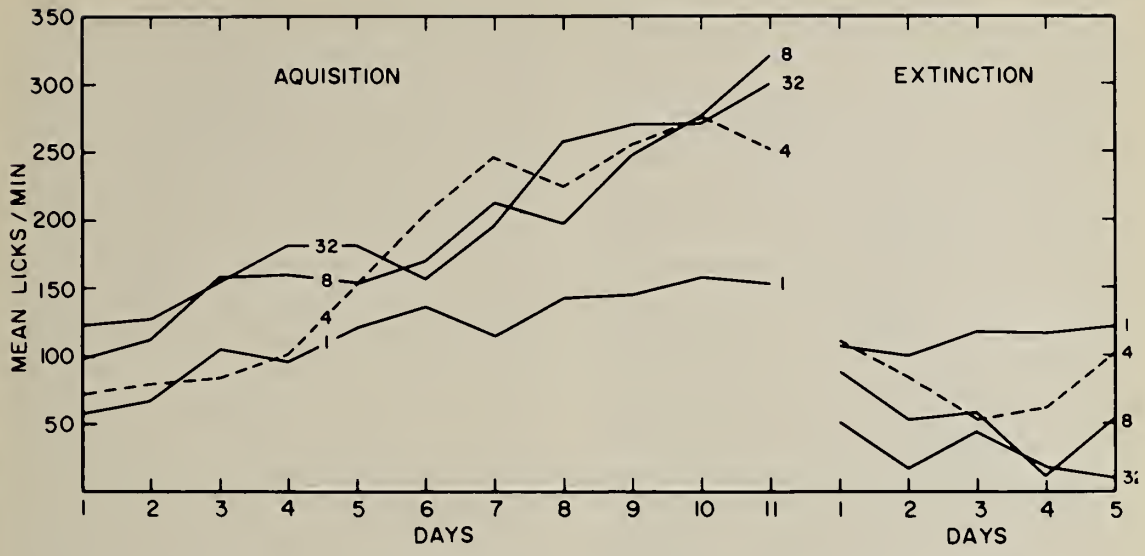


Figure 29–12. Levels of tongue activation as related to concentration of sucrose solutions during successive stages of habituation. The curves show mean licks per minute during acquisition and extinction. *From data of* Dr. C. L. Trafton, reported by Young (1966).

of primary motivation: a vertical dimension of *activation* and a horizontal dimension of *hedonic value.*

Affective processes are both activating and directive. They are directive in the sense that they are either GO or STOP. They regulate behavior as traffic lights regulate the flow of traffic. Affective processes are also reinforcing in the sense that they lead to repetition of activities through which animals learn. Learning and hedonic motivation are closely interrelated processes of behavioral development.

We have examined the role of affective processes in the development of sweet taste preferences. I wish now to consider some of the broader implications of hedonic theory.

The utility of pleasantness and unpleasantness

In an important series of experiments Cabanac has shown that a stimulus can arouse pleasant or unpleasant feelings depending upon its usefulness in maintaining homeostasis.[1] He made a series of experiments with tastes, odors, and temperatures. Let us consider his work with temperatures.

In the thermal experiments the subject was immersed in a stirred water bath while making affective reports after a 30-second thermal stimulation of the left hand. Cabanac found that responses differed according to the thermal state of the subject. When the internal body temperature was high (hyperthermic), the cold or cool stimuli were reported as pleasant and the warm or hot stimuli as unpleasant. When the internal body temperature was low (hypothermic), the cold or cool stimuli were unpleasant and warm or hot stimuli were pleasant. The temperatures of the bath (15° and 45° C) were between the lower

and upper thresholds of pain; so pain was not a factor in the hedonic response.

Cabanac demonstrated that a specific nonpainful thermal stimulus is perceived as either pleasant or unpleasant according to the internal body temperature of the subject. A hypothermic subject will *like* a hot, even slightly burning, stimulation. A hyperthermic subject will *like* a biting cold stimulus even if the temperature is slightly painful. Therefore, Cabanac argued, pleasure occurs when sensation indicates the presence of a stimulus which helps to maintain homothermia—a relatively constant internal body temperature which for man is about 37° C. The affective arousals are clearly useful when considered in relation to the homeostasis of body temperature.

Cabanac's subjects could easily distinguish between the sensory and hedonic aspects of experience.

In other experiments Cabanac found that sweet-tasting sucrose and orange odor were pleasant when the subjects fasted, but the gustative and olfactive stimuli were unpleasant after the subjects had ingested 100 grams of glucose in water. The internal signals that produced hedonic changes during deprivation and satiation are unknown.

Homeostasis and taste

In defending a dubious hypothesis, J. A. Deutsch and A. D. Jones presented some data that are of interest quite apart from their theory.[2] Preference tests were made with two methods: (1) The familiar T maze was used with the incentives out of range of the head receptors at the point of choice. (2) A testing cage was used with the incentives relatively close together during a 20–minute exposure.

All tests were made with two saline solutions with concentrations of 0.2% NaCl and 0.8% NaCl. Other studies have shown that a saline concentration of 0.8% is nearly optimal with rats. In fact, Nelson in a brief report stated that rats ingest excessive quantities of NaCl when the concentration is 0.8%; their growth is retarded, kidneys and other organs become enlarged and there are other symptoms of excessive intake.[7]

Deutsch and Jones made all tests with a 20-hour deprivation of water. We may assume that the need of the animals for water was approximately constant during all preference tests. Under these conditions the two methods of testing preferences yielded different results. With the T maze technique the rats steadily learned to select the *weaker* saline solution (0.2% NaCl>0.8% NaCl). With the continuous choice method of testing, the rats showed a clear preference for the *higher* concentration (0.8% NaCl>0.2% NaCl). The choice with the T maze technique is wise and adaptive for it is generally known that the ingestion of saline solutions tends to heighten thirst rather than to relieve it. A choice of water would be still better. The choice with the continuous exposure technique is also wise and adaptive but in a different way. The animal must somehow locate and ingest salt in order to maintain a homeostasis of sodium in the blood serum. A concentration of 0.8% NaCl is very near to the concentration in the blood serum.

These data are interesting because they demonstrate the existence of

separate bodily mechanisms that control the intake of foods. The data are interesting, also, from the methodological point of view. They clearly show that the T maze is not the best method for observing preferences based on palatability. The T maze, however, may be serviceable in studies of learning under conditions of metabolic and nutritional need.

Conclusion

In concluding this theoretical discussion I would like to speculate a bit regarding the kind of neurochemical mechanism that is required to explain the facts.

We must postulate the existence within the brain of a mechanism that evaluates and integrates stimulations from the different modalities. We know that acceptance and rejection are *evaluative* reactions that are equivalent to the judgments *good* or *bad*. Some stimulations are hedonically positive; others are negative. We know that positive and negative affective arousals are integrated according to precise laws of algebraic summation. And we know that appetitive and aversive behavior varies with the internal chemical and nutritional state of the organism. Also we know that the positive and negative reactions are fairly uniform within a given species. These facts require an explanation.

We postulate the existence of adaptive mechanisms that have evolved through millions of years of organic evolution. Ethologists have described instinctive reactions that relate to defense and aggression, to obtaining food, to reproduction, care of the young, and other basic matters. Instinctive reactions are elicited by specific patterns of stimulation. The reaction to a sweet taste is just one specific instance of a more general biological principle.

The sensitivity to sweet is biologically adaptive; it aids in recognition and selection of carbohydrates that are necessary as a source of energy. Ripe fruits are sweet and preferred to green sour fruits which are unpleasant. Bitter tastes are unpleasant; they indicate the presence of toxic alkaloids. Salty tastes indicate the presence of sodium which is required to maintain the homeostatic environment of the cells. The biological utility of these reactions is obvious. Chemical, thermal and tactual receptors at the oral end of the alimentary canal stand guard over the acceptance or rejection of foodstuffs.

We postulate that the receptors which yield hedonic responses to specific kinds of stimuli, feed neural excitations into a nonspecific neural structure where facilitative and inhibitory excitations are integrated.

We have seen that hedonic processes are both activating and reinforcing. I will add that hedonic processes are also *informational.* The information indicates the way in which a stimulus-situation *affects* the organism—positively or negatively. This is important information.

Human beings are definitely aware of the pleasures and displeasures of sensation. The affective and cognitive processes occur at different psychological levels of functioning. The cognitive processes, presumably, are mediated at the level of the cerebral cortex. Affective processes are initiated by subcortical mechanisms.

The relation between affective and cognitive processes is twofold. We are

aware that pleasantness and unpleasantness occur. We are also aware that perception and memory have a powerful influence upon our moods of cheerfulness and depression. Consider, for example, the effects of good news and bad news, of victory and defeat, of success and failure. Consider also the fact that cognitive evaluations reinforce and direct the course of learning. Words like *good* or *bad*, *right* or *wrong, correct* or *incorrect,* influence feelings and have a directive influence on performance.

The investigation of the interrelations between affective and cognitive processes should have high priority as an area for psychological research.

We live in a world defined by perceptions, memories, imaginations, feelings, emotions, beliefs and desires, rather than in a world defined by stimulus and response. This is the individual's world of subjective experience. We recognize that in this world man is partly a creature of reason and partly a creature dominated by emotion.

REFERENCES

1. Cabanac, M.: Physiological role of pleasure. *Science,* 173: 1103–1107, 17 September, 1971.
2. Deutsch, J.A., and Jones, A.D.: Diluted water: An explanation of the rat's preference for saline. *J. comp. physiol. Psychol.* 53: 122–127, 1960.
3. Dixon, W.J., and Massey, F.J., Jr.:, *Introduction to Statistical Analysis.* (2nd ed.) New York: McGraw-Hill, 1957.
4. Guilford, J.P.: System in the relationships of affective value to frequency and intensity of auditory stimuli. *Amer. J. Psychol.,* 67: 691–695, 1954.
5. Harris, L.J., Clay, J., Hargreaves, F.J., and Ward, A.: Appetite and choice of diet: The ability of the vitamin B deficient rat to discriminate between diets containing and lacking the vitamin. *Proc. Roy. Soc., Series B (biological sciences),* 113: 161–190, 1933.
6. Henle, M.: E. B. Titchener and the case of the missing element. *J. Hist. behav. Sci.,* Vol. X, No. 2: 227–237, April 1974.
7. Nelson, D.: Do rats select more sodium chloride than they need? *Fed. Proc. amer. physiol. Soc.,* 6, Whole No. 169, 1947.
8. Pfaffmann, C.: The pleasures of sensation. *Psychol. Rev.* 67: 253–268, 1960.
9. Richter, C.P.: Total self regulatory functions in animals and human beings. In *The Harvey Lecture Series,* Vol. 38, 1942, pp. 63–103.
10. Richter, C.P.: The self-selection of diets. In *Essays in Biology, in honor of Herbert M. Evans,* Univ. of Calif. Press, 1943, pp. 501–505.
11. Richter, C.P., and Campbell, K.H.: Taste thresholds and taste preferences of rats for five common sugars. *J. Nutrition* 20: 31–46, 1940.
12. Young, P.T.: Relative food preferences of the white rat, II. *J. comp. Psychol.,* Vol. XV: 149–165, 1933.
13. Young, P.T.: Studies in food preference, appetite and dietary habit, V. Techniques for testing food preference and the significance of results obtained with different methods. *Comparative Psychology Monographs,* 1945, 19, pp. 1–58.
14. Young, P.T.: Studies of food preference, appetite and dietary habit, VII. Palatability in relation to learning and performance. *J. comp. physiol. Psychol.,* 40: 37–72, 1947.
15. Young, P.T.: The role of affective processes in learning and motivation. *Psychol. Rev.,* 66: 104–125, 1959.
16. Young, P.T.: Hedonic organization and regulation of behavior. *Psychol. Rev.* 73: 59–86, 1966. (Reprinted as P–710 by Bobbs-Merrill Co., 4300 W. 62nd St., Indianapolis, Ind.)
17. Young, P.T.: Palatability: The hedonic response to foodstuffs. In *Handbook of Physiology, Section 6, Alimentary Canal.* Washington, D. C. American

Physiological Society, 1967, pp. 353–366.

18. Young, P.T.: Evaluation and preference in behavioral development. *Psychol. Rev.:* 75, 222–241, 1968.
19. Young, P.T.: *Emotion in Man and Animal: Its Nature and Dynamic Basis.* 2nd revised ed., Huntington, New York, Krieger Publishing Co., 1973.
20. Young, P.T., and Chaplin, J.P.: Studies in food preference, appetite and dietary habit, III. Palatability and appetite in relation to bodily need. *Comparative Psychology Monographs,* 1945, 18, pp. 1–45.
21. Young, P.T., and Madsen, C.H., Jr.: Individual isohedons in sucrose-sodium chloride and sucrose-saccharin gustatory areas. *J. comp. Physiol. Psychol.,* 56: 903–909, 1963.

Author Index

Pages on which presentations by conference participants appear are set in bold face type. Numbers in italics indicate pages on which bibliographic references appear and immediately follow the page numbers of text citation.

Adair, E.R., 301, *306,* 308, *308* 320, *324*
Adams, L.A., 86, *97*
Adler, J., 187, *187*
Adolph, E.F., 249, 263, 331, 333, 335, *343*
Agarwal, J., 210, *212*
Ahokar, A., 319, *325*
Ahrens, R.A., 374, 384, *385*
Aiyar, R., 210, *212*
Akazawa, N., 262, *264*
Akesson, C., 167, *172*
Alexander, D.P., 61, *62*
Allen, R.P., 384, *385*
Alterman, A.I., 384, *386*
Amenomori, Y., 80, *83*
Andersen, H.T., 9, *19*
Anderson, T.A., 235, *244*
Andrews, K., 67, *67,* 162, 163, *172*
Anova, D.D., 237, *244*
Anteby, S.C., 61, *62*
Arai, M., 250, *264*
Arballo, J.C., 120, *123*
Arsi, M., 250, *264*
Astrom, K.E., 62, *62*
Atema, J., 17, 18, *19*
Aukrovd, W.R., 244, *244*

Bacon, W.E., 357, *361*
Balch, C.C., 249, *263*
Baldwin, B.A., 90, 92, *98*
Balint, M., 147, *157*
Ball, G., 310, *325*
Baradi, A.F., 71, *83*
Bard, P., 66, *66,* 194, *202*
Barelare, B., 249, *265*
Barnes, R.H., 93, *98*
Barnett, S.A., 218, 225, *231,* 249, *263*
Barth, E.E., 58, *62*
Bartolovic, B., 320, *324,* 328
Bartoshuk, L., 11, 13, *19,* 13, *21,* 26, 27, *39,* 124, *124,* 156, *157*
Battaglia, F.C., 61, *63*
Bavly, S., 241, *244*
Beatty, W.W., 310, *324*
Becker, E.E., 310, *324*
Becker, R.R., 58, *62*
Beebe-Center, J.G., 289, *294*
Behar, M., 237, 238, *244*
Beidler, L.M., 5, 6, *21,* 54, *62,* 70, 74, *83,* 124, *124*
Bell, R.H.V., 331, *343*
Bell R.R., 243, *244.*
Belluzzi, J.D., 295, *307*
Benjamin, R.M., 14, 19, *20*
Bennett, F.J. 242, *245*
Bergman, J.G., 243, *244*
Berman, S.N., 235, *245*
Bernard, R.A., 8, *21,* 42, *46*
Bhagavan, H.N., 43, 46, *46*
Bibby, B.G., 345, *348*
Bicchieri, M.G., 4, *19*
Biester, A., 388, *397*
Bigelow, G., 384, *385,* 384, *386*
Bines, J.A., 249, *263*
Blaney, J.R., 347, *349*
Blanton, M.G., 126, *139*
Boddy, K., 111, *122*
Bolles, R., 310, 319, *325,* 335, 338, 341, *343,* 357, 359, *361*
Bonow, B.E., 346, *348,* 351
Book, S.A., 86, *97*
Booth, D.A., 358, 359, *361*
Borer, K., 18, *19*
Borison, H.L., 190, *202*
Bosack, T.N., 147, *157,* 146, *158*
Bosma, J.F., 111, *122*
Bourne, G.H., 71, *83*
Bowden, C.L., 384, *385*
Bradley, R.M., 52, 58, *62,* 58, *63,* 102, *107,* 114, *122,* 111, *123,* **51-64**
Branch, L., 269, *279*
Bratthall, D., 347, *348*
Braun, J.J., 200, *202*
Bray, G.A., 295, *306*
Breese, G.R.,16, *21*
Brightman, V.J., 184, *187*
Brobeck, J.R. 22, *22*
Brookshire, K.H., 43, 46, *46*
Brown, E., 42, *46*
Brown, J., 146, 147, *157*
Bruce, R.H., 234, *234*
Brunelle, J.A., 348, *349*
Bryan, E.S., 128, *139*
Burke, P., 141, *141*
Burns, M.P., 86, *97*
Burton, H., 14, 19, *20*
Bustad, L.K., 86, *97*
Butcher, M.J., 135, 137, *140*
Butler, D.G., 90, *98*

Cabanac, M., 292, *294*, 297, 298, 301, *306*, 308, *308*, 320, *324*, 370, 372, *372*, 413, *416*
Cagen, R.H., 310, *324*
Calloway, J.W., 93, *98*
Cameron, A.T., 9, *20*, 101, *107*
Campbell, B.A., 95, *98*
Campbell, B.E., 357, *361*
Campbell, D., 133, *139*, 146, 147, *157*
Campbell, H.K., 364, 372, *373*
Campbell, J., 90, *98*
Campbell, K.H., 9, *20*, 42, *46*, 387, *397*, 406, *416*
Campbell, R.G., 347, *348*
Canestrini, S., 127, *139*
Capretta, P.J., 357, 359, *361*
Carol, R., 235, *245*
Carroll, N.V., 243, *245*
Chan, J.C.M., 235, *245*, 249, *263*
Chaplin, J.P., 249, *265*, 409, *417*
Chapman, M.J., 156, *158*
Chappell, G., 163, *172*, 283, *294*
Chase, H.P., 88, *97*
Chen, C.L., 80, *83*
Childs, G.M., 238, *244*
Chrisp, R.B., 269, *279*
Christiansen, J.H., 331, *344*
Christiansen, R.L., 205, *212*
Clark, D.M., 58, *62*
Clark, M.M., 218, 223, 224, 225, 228, *231*, 234
Clay, J., 411, *416*
Cohen, A.M., 241, *244*
Cohen, L.B., 209, *212*
Cohen, M., 384, *385*
Collazos, C., 237, *245*
Collier, G., 310, 319, *325*, 342, *342*, 333, 335, 337, 338, 340, 341, 342, *343*, 339, 342, *344*, 357, 359, *361*, **23, 47, 64, 141, 159, 247, 281, 330-344**
Collings, V., 205, *212*
Combs, G.E., 93, *98*
Committee on Nutrition: 249, *263*
Conel, J.L., 62, *62*, 62, *63*
Cooper, B.R., 16, *21*
Corbit, J., 165, *172*, 331, *343*
Corbit, J.D., 191, *202*, 295, *306*, 309, 312, *325*, 357, *361*
Corbit, T.E., 144, *145*
Cordero Funes, J.C., 120, *123*
Cori, C.F., 388, *397*
Cornelison, F.S. 384, *386*
Cowell, C., 235, *245*
Cox, V.C., 48, *48*
Crawford, H.D., 384, *386*
Crook, C., 107, *107*, 210, *210*, **146-157, 158, 159, 160**
Cruce, J.A.F., 313, *325*

Dabiere, C.S., 88, *97*
d'Aquili, F., 248, *248*
Davies, E., 174, *188*
Davies, J.N.P., 239, *244*
Davis, C.M., 249, *263*
Davis, D.D., 192, *202*
Davis, J.D., 359, *361*
Davis, M.E., 61, *63*
Dawes, G.S., 58, 61, *63*, 111, *112*
de Barrios, G., 239, *245*
Deerr, N., 4, *20*
DeLucia, C.A., 132, *139*, 146, 148, *157*
DeSnoo, K., 60, *63*
Desor, J.A., 9, *20*, 51, *63*, 67, *67*, 91, *97*, 107, *107*, 131, *140*, 143, *145*, 148, 155, *157*, 161-168, 170, *172*, 235, 236, *245*, 298, *306*, **67, 161-172**
Dethier, V.G., 14, *20*, 187, *187*, 213, *213*, 364, *372*
Deutsch, J.A., 414, *416*
Dews, P.B., 52, *63*
Dhariwal, A.P.S., 79, 80, *84*
Diamant, H., 7, 9, *20*
Dickens, D., 249, *263*
Dixon, W.J., 405, *416*
Doetsch, G.S., 35, *39*
Donnelly, C.J., 347, *349*
Draper, H.H., 243, *244*
Drier, T., 146, *157*
Drummond, M., 127, 128, *139*
Dubignon, J., 133, *139*, 147, *157*
DuBose, C.N., **99-107**
Duclaux, R., 297, 298, 301, *306*, 308, *308*
Duffy, M., 357, 359, *362*
Duggan, S.Z., 306, *307*, 315, *325*
Dunham, R.B., 306, *307*, 315, *326*

Easterday, C.L., 61, *63*
Eckstein, A., 128, *139*
Edelman, J., 241, *246*
Edelstein, E., 174, *188*
Eeg-Larsen, N., 345, *348*
Eglitis, J.A., 114, *123*
Eibl-Eibesfeldt, I., 186, *187*
Eindhoven, J., 269, 270, *279*
Ekman, G., 167, *172*
Ellison, G.D., 16, *21*
Engel, R., 282, 283, *294*
Engen, T., 107, *107*, 143, 144, *145*, 148, *157*, 210, *212*, 290, *294*, **143-145, 279, 327**
Englander, H.R., 348, *349*
Eppright, E.S., 240, *244*
Epstein, A.N., 93, *97*, 95, *98*, 331, *344*
Erickson, R.P., 14, *20*, 26, 35, *39*
Ernits, T., 165, *172*, 357, *361*
Ervin, F.R., 359, *361*
Ervin, R.F., 347, *349*
Erwin, W.G., 347, *349*
Estes, R.D., 331, *343*

Faillace, L.A., 384, *385*
Falk, J.L., 357, *362*, 374-377, *385*, 377, 378, 381, *386*, **374-386**
Falkenberg, P., 35, *39*
Farbman, A.I., 52, *63*, 70, 71, 73, *83*, 85, *85*,
Ficken, M., 171, *172*
Filer, L.J., 235, *244*
Filion, R.D.L., 248, *248*

Finnegan, L., 178, *188*
Fish, H.S., 7, *21,* 71, 73, *83*
Fisher, G.L., 7, 9, 10, *20,* 42, *46,* 198, 199, *202,* 111, *122*
Fisher, R., 111, *122*
Fitzgerald, R.J., 348, *349*
Foll, C.V., 238, *244*
Fomon, S.J., 235, 240, *244*
Foster, R., 92, *97*
Fox, H.E., 58, *63,* 111, *122*
Fox, H.M., 240, *244*
Fox, S., 248, *248*
Frank, M., 7, *20,* 13, *21,* 26-31, 33-35, *39,* 198, 199, *202,* **25-39, 40, 41**
Freedman, D.G., 186, *187*
Frings, H., 3, *20*
Frostell, G., 346, 347, *348*
Fryer, B.A., 240, *244*
Fuller, E.S., 240, *244*
Fuller, J.L., 295, *306,* 357, *361*
Funakoshi, M., 9, *19,* 7, 9, *20*

Galanter, E., 284, *294*
Galef, B.G., 51, *63,* 71, *83,* 218, 223-225, 227-230, *231,* 232, 233, 234, **68, 217-232, 233, 234, 280**
Ganchrow, J., 9, 10, *20*
Garcia, J., 19, *20,* 218, *231,* 359, *361*
Garcia, L., 239, *245*
Gasparian, R.E., 144, *145*
Gautier, E., 160, *160*
Gedelia, I., 61, *62*
Glauser, E.M., 86, *97*
Goddard, K.F., *158*
Goetzl, F., 319, *325*
Gold, R.M., 15, *20*
Goldblatt, P.B., 248, *248*
Goldman, R., 314, *326*
Goldrick, B., 280, *280,* 323, *325*
Goldrick, N., 213, *213*
Gonzalez, N.L., 243, *244*
Gordon, A., 314, *326*
Gordon, H.A., 347, *349*
Gordon, R.P., 248, *248*
Goss, B.J., 347, 348, *349,* 351
Gottheil, E., 384, *386*
Graff, H., 297, *306,* 310, *325*
Graham, R., 88, *98*
Grahnew, H., 346, *348,* 351
Greene, J.T., 42, *46,* 357, *362*
Greene, L., 107, *107,* 167, 168, 170, *172,* **161-172**
Greenwood, M.M., 135, 137, *140*
Greenwood, M.R.C., 317, 322, *325,* 313, *326*
Gresham, E.L., 61, *63*
Griffiths, R., 384, *385,* 384, *386*
Grinker, J., 291, *294,* 298, *306,* 303, *307,* 309, 310, 313, 315, 317, 320, 322, *325,* 310, 313, 315, *326,* **40, 68, 248, 280, 309-327, 328, 329**
Gross, W., 320, *326*
Grossi, M., 235, *245*
Grossman, J.D., 87, *98*
Grossman, S.P., 295, *307,* 309, *326*
Grosvenor, C.E., 79, 80, *84*
Guggenheim, B., 348, *349,* 351
Guggenheim, K., 323, *325*
Guilford, J.P., 405, *416*
Gunther, M., 138, *139*
Gurwitz, S., 143, *145,* 148, *158,* 162, *172,* 235, 236, *245,* 298, *307*
Gustafsson, B.F., 346, *348,* 351
Guth, L., 70, *84*
Guttman, N., 11, *20,* 42, *46,* 357, *361*

Hagstrom, E.C., 309, *326,* 357, *361,* 366, 372
Haith, M.M., 146, *157,* 209, *212*
Halbreich, U., 174, *187*
Hall, K.R.L., 217, *231*
Halpern, P.B., 8, *21,* 42-45, *46,* 47, 48
Hamilton, C., 171, *172*
Hamilton, C.L., 22, *22,* 249, *263,* 331, *343*
Hamilton, J.R., 90, *98*
Hamilton, L.W., 341, *344,* 384, *386*
Hamlin, P., 342, *343*
Hankins, W.G., 19, *20*
Hara, M., 250, 254, 260, *264*
Hardwick, J.L., 345, *349*
Hargreaves, F.J., 411, *416*
Harrell, E.H., 42, *46*
Harriman, A.E., 372, *372*
Harris, L.E., 235, *244,* 249, *263*
Harris, L.J., 411, *416*
Harrison, R.W., 347, *349*
Hasenzahl, B., 285, *294*
Hashim, S.A., 295, *306,* 314, *325*
Heiber, L., 229, *231,* 232
Henderson, P.W., 71, *83,* 227, *231,* 233, **70-83, 85**
Henkin, R.I., 205, *212*
Herman, C.P., 298, *306,* 305, *307*
Herrick, C.J., 17, 18, *20,* 192, *202*
Hertz, D., 174, *187*
Hester, H.R., 88, *98*
Hidaka, I., 26, *39*
Hill, S., 165, *172*
Hirsch, E., 332, *332,* 342, *342,* 342, *343,* 342, *344,* **330-334**
Hirsch, J., 280, *280,* 291, *294,* 298, *306,* 309, 313, 317, 323, *325*
Hitchcock, J.T., 237, *245*
Hoebel, B.G., 249, *263*
Holt, L.F., 249, *265*
Honigschmied, J., 70, *84*
Hooker, D., 186, *187*
Hoppert, C.A., 347, *349*
Hoshino, K., 250, *264*
Hossain, M.A., 244, *244*
Hough, L., 241, *246*
Houpt, K.A., 95, *98,* **86-98**
Houpt, T.R., 93, *98,* **86-98**
Hsiao, S., 42, *46,* 357, *361*
Hubel, D.H., 61, *63*
Huenemann, R.L., 237, *245*
Hulse, S., 357, *360*

Humaraith, V., 285, 292, *294*
Humphrey, T., 186, *187*
Hunt, H.R., 347, *349*
Huxley, H.G., 346, *349*

Iwasaki, S., 249, *264*

Jacobs, H.L., 3, *20*, 22, *22*, 101, 102, 106, *107*, 269, *279*, 285, 289, 290, 292, *294*, 318, *326*, 336, 337, *344*, 357, 359, *361*, **21, 47, 99-107, 233, 247, 350**
Jacoby, G.A., 295, *306*
Jasper, H.H., 209, *212*
Jeffrey, W.E., 209, *212*
Jelliffe, D.B., 238, 239, 242, *245*
Jelliffe, E.F.P., 239, 242, *245*
Jennings, H.S., 187, *188*
Jensen, K., 126, 129, *140*, 210, *212*
Jessup, A., **42-47**
Jerome, N.W., 235, 240, *245*, **235-246, 247, 248, 327**
Johnson, P., 107, *107*, 113, *122*, 113, 115, 120, *123*, **64, 111-123, 141**
Johnson, P.R., 313, *325*
Jolly, A., 4, *20*
Jones, A.D., 414, *416*
Jones, M.R., 392, *397*
Jordan, H.A., 166, 170, *172*, 306, *307*, 315, *325*, 369, *372*
Jordan, H.V., 347, *349*
Jump, E.B., 88, *98*

Kakolewski, J.W., 43, 46, *46*, 48, *48*
Kalat, J.W., 43, 45, *46*, 62, *63*, 218, *232*
Kalmus, H., 173, *188*
Kamen, J.M., 269, 270, *279*
Kanarek, R.B., 331, 342, 343, *344*
Kanouse, D.E., 315, *326*
Kare, M.R., 3, *20*, 8, *21*, 42, *46*, 90, *98*, 171, *172*
Kashiwabara, S., 249, *264*
Kato, M., 252, 260, *264*
Kaufman, N.A., 323, *325*
Kawamura, Y., 194, *202*
Kaye, H., 133, *140*, 142, *142*, 147, *157*, 146, *158*
Keen, H., 323, *325*
Keen, R., 146, *157*
Keesey, R.E., 249, *264*
Kelly, M., 90, *98*
Kennedy, G.C., 309, *325*
Kennedy, J.M., 90, 92, *98*
Kessen, W., 195, 197, *202*, 209, *212*
Keyes, P.H., 346, 347, *348*, 347, *349*
Kiser, B.B., 235, 240, *245*
Kissileff, H.R., 310, *324*
Kluter, R.A., 269, *279*, 289, 290, *294*, 318, *326*
Knittle, J.L., 298, *306*, 313, *325*
Kobayashi, Y., 249, 254, *264*
Kobre, K.R., 132, 134, 138, *140*, 143, 144, *145*, 148, 155, *157*
Koch, C., 315, *326*
Koelling, R.A., 218, *231*
Kolata, G.B., 5, *20*
Konig, K.G., 348, *349*, 351
Konuma, Y., 249, *264*
Krasse, B., 346, *348*, 351
Kruk, H., 331, *344*
Kohn, M., 42, *46*
Krasse, B., 346, *349*
Krauss, R.M., 249, *263*
Kreshover, S.J., **XV**
Kron, R.E., 148, *158*
Kroner, T., 127, *140*
Kual, K.K., 237, *244*
Kulvinskas, C., 61, *63*
Kupfermann, I., 249, *263*
Kussmaul, A., 126, *140*
Kuwabara, K., 252, *264*

Lalonde, E.R., 114, *123*
Lamkin, G.H., 240, *244*
Larje, O., 346, *349*
Larkin, R., 310, *325*
Larson, R.H., 346, 347, *348*, 346, 347, 348, *349*, 351, **345-350, 351**
Lât, J., 333, 336, *344*
Lawrence, D.H., 358, *362*
Lawson, D., 156, *158*
Leavitt, M.D., **XIII, XIV**
Leduc, B.M., 58, *63*, 111, *122*
Lee, R.B., 242, *245*
Le Magnen, J., 19, *20*, 51, *63*, 107, *107*, 174, *188*, 233, *233*, 306, *307*, 357, 358, 359, *361*, **21, 40, 142, 203, 233, 308, 329, 355-362**
Leon, M., 81, *84*, 229, *231*
Leshner, A.I., 333, 336, 339, *343*, 338, 339, *344*
Lev, R., 58, *63*
Levin, B.B., 242, *245*
Levin, G.R., 142, *142*
LeVine, R.A., 242, *245*
Levy, H.L., 61, *63*
Liebson, I.A., 384, *385*, 384, *386*
Liggins, G.C., 58, *63*, 111, *122*
Liley, A.W., 60, 61, *63*
Linker, E., 319, *326*
Lipsitt, L.P., 107, *107*, 120, *123*, 127, 132, 135, 137, 138, *140*, 143, 144, *145*, 148, *157*, 146, 155, *158*, **65, 125-140, 141, 142**
Littlefield, J.W., 61, *63*
Leonard, C., 14, *20*
Lovett, D., 358, 359, *361*
Lundqvist, C., 346, *348*, 351
Luther, E.C., 120, *123*
Lytle, L.D., 95, *98*

Macht, M.B., 18, *20*, 66, *66*, 194, *202*
Mackay, E.M., 93, *98*
Macy, I.G., 249, *263*
Maddux, J.F., 384, *385*
Madsen, C.H., Jr., 404, 406, 407, *417*
Maiweg, H., 80, *84*
Makowski, E.L., 61, *63*
Malone, P.D., 7, *21*, 71, 73, *83*

Maller, O., 9, *20*, 51, *63*, 67, *67*, 91, *97*, 107, *107*, 131, *140*, 143, *145*, 148, 155, *157*, 161-168, 171, *172*, 174, *188*, 235, 236, *245*, 298, *306*, 310, *324*, 310, *326*, **141, 161-172**
Mamanouchi, A., 235, 260, *264*
Marlow, R.A., 88, *97*
Marshall, D.A., 35, *39*
Marshall, J.F., 16, *20*
Marshall, N.B., 331, *344*
Marthaler, T.M., 345, 346, *349*
Martinez, J.A., 235, *244*
Masayekhi, M.B., 331, *344*
Maslansky, F., 235, *245*
Mason, D.J., 42, *46*
Massey, F.J., Jr., 405, *416*
Masuoka, D., 16, *21*
Matsushima, T., 249, *264*
May, R.M., 70, *84*
Mayer, J., 42, *46*, 97, *98*, 249, *263*, 320, *326*, 331, *344*
Mead, M., 237, *245*
Meiselman, H.L., 101, *107*, 269, *279*, 285, *294*, **269-279, 280, 281**
Meites, J., 80, *83*
Mellberg, J.R., 348, *349*
Mello, N.K., 375, 384, *386*
Mellor, D.J., 61, *63*
Mena, F., 79, 80, *84*
Menaker, L., 249, *263*
Mendel, L.B., 392, *397*
Mendelson, J.H., 384, *386*
Merry, J., 384, *386*
Mersman, H.J., 88, *98*
Meschia, G., 61, *63*
Metropolitan Life Insurance Company, 277, 278, *279*, 295, *307*
Miyahara, C., 249, 258-261, 263, *264*, 332, 336, *344*
Miller, F.R., 65, *65*, 193, *202*
Miller, N.E., 42, *46*
Miller, W.D., 345, *349*, 350
Minaire, Y., 301, *306*, 308, *308*, 320, *324*
Minturn, L., 237, *245*
Mistretta, C.M., 39, *39*, 52, 58, *62*, *52*, *58*, *63*, 70, 73, *84*, 85, *85*, 102, *107*, 111, 114, *122*, 111, *123*, **39, 47, 51-64, 65, 66, 67, 68, 69, 85, 123, 248**
Mizuno, K., 254, *264*
Moltz, H., 81, *84*, 229, *231*
Moncrieff, R.W., 188, *188*
Montag, P.P., 61, *63*
Montagu, M.F.A., 126, *140*
Mook, D.G., 357, *361*, 365, *373*
Moorcroft, W.H., 95, *98*
Moore, M.E., 248, *248*, 319, *326*
Morimoto, T., 194, *202*
Morrison, G.R., 43, *46*, 205, 210, *212*, **42-47, 48, 279**
Moskowitz, H.R., 9, *20*, 101, *107*, 161, *172*, 197, *202*, 269, *279*, 284, 285, 287, 289, 290, 291, *294*, 299, *307*, 318, *326*, **188, 246, 247, 282-294, 327**
Moskowitz, L., 144, *145*
Mount, L.E., 89, *98*
Mouratoff, G.J., 243, *245*
Mrosovsky, N., 295, *307*
Mühlemann, H., **350**
Mukherjee, P.S., 238, *245*
Murata, H., 249, 260, *263*, 249, *265*
Murphy, B.F., 384, *386*
Murray, E.J., 42, *46*
Muto, S., 249, 262, *263*, 249, 250, 252, 253, 254, 258-261, 263, *264*, 249, 260, *265*, 332, 336, *344*, **249-265**
Myers, R.D., 375, 380, *386*

Nachman, M., 51, *63*, 310, *326*, 359, *361*, 372, *373*
Naf, D., 160, *160*
Naito, J., 249, *263*, 253, 260, *264*, 249, 260, *265*
Navarick, D.J., 341, *341*, 384, *386*
Navis, J.M., 249, *263*
Neligan, G.A., 88, *98*
Nelson, A.K., 126, 128, *140*
Nelson, D., 414, *416*
Nelson, T., 61, *64*
Nisbett, R., 143, *145*, 148, *158*, 162, *172*, 191, *202*, 235, 236, *245*, 295-298, 301, 303, 304, *307*, 314, 315, *326*
Nixon, D.A., 61, *62*
Nordsiek, F.W., 309, *326*
Norgren, R., 14, *20*
Norrison, W., 205, 210, *212*
Nowlis, G.H., 48, *48*, 66, *66*, 107, *107*, 131, *140*, 159, *159*, 190, 195, 197, 200, *202*, 210, *212*, **48, 65, 159, 190-203, 204, 232, 246**

Oakley, B., 70, 71, *84*
O'Conner, M., 348, *350*
Ogawa, H., 26, *39*
Olmsted, J.M.D., 70, *84*
Opsahl, C.A., 22, *22*
Orland, F.J., 347, *349*
Orlic, D., 58, *63*
Ouellet, J.V., 200, *202*
Ough, C.S., 269, *279*
Owen, G., 241, *245*

Pangborn, R.M., 167, *172*, 269, *279*, 287, *294*, 323, *326*
Panksepp, J., 331, *344*
Patwardhan, V.N., 238, *245*
Payne, J., 319, *325*
Peck, M.B., 143, 144, *145*, 148, *157*
Peiper, A., 126, *140*
Pekas, J.C., 93, *98*
Peryam, D.R., 269, 270, *279*, 285, *294*
Peterson, F., 126, 127, *140*
Pfaffmann, C., 7, 8, 9, 10, 12, 13, 14, *20*, *21*, 22, *22*, 26, 27, *39*, 42, *46*, 51, *64*, 99, 106, *107*, 138, *140*, 156, *157*, 174, *188*, 194, 198, 199, *202*, 205, *212*, 233, *233*, 309, 317-319, *326*, 333, *344*, 357, *361*, 366, *372*, **3-21, 22, 23, 24, 40, 41, 65, 68, 123, 189, 212, 213, 248, 308, 350**
Phoenix, M.D., 148, *158*
Pierson, A., 174, *188*
Pilgrim, F.J., 269, 270, *279*, 285, *294*
Pilleri, G., 187, *188*
Pizanti, S., 174, *187*
Polemis, B.W., 269, 270, *279*

Polivy, J., 298, *306*
Pond, W.G., 90, *98*
Potter, E.L., 61, *63*
Powley, T.L., 22, *22,* 249, *264*
Pozmanski, R., 241, *244,* 323, *325*
Prader, A., 160, *160*
Pratt, K.C., 126, 128, *140*
Preyer, W., 126, *140*
Price, J.M., 303, *307,* 317, 322, *325,* 315, *326*
Printer, S., 111, *122*
Prior, I.A.M., 243, *245*
Pritchard, J.A., 58, 59, *63*
Purvis, G., 235, *245*

Quartermain, D., 313, *325*
Quensel, C.E., 346, *348,* 351

Rainey, L.H., 126, 127, *140*
Rankin, J.H.G., 61, *63*
Reed, T., 269, *279*
Rao, K.S., 238, *245*
Reichel-Dolmatoff, A., 237, *245*
Reichel-Dolmatoff, G., 237, *245*
Reilly, B.M., 135, 137, *140*
Remley, N.R., 42, *46*
Resnick, J.B., 347, *350*
Reyniers, J.A., 347, *349*
Rice, K.K., 395, *398*
Richards, R.T., 58, *63,* 111, *122*
Richter, C.P., 9, *20,* 7, *21,* 42, *46,* 71, 73, *83,* 106, *107,* 198, *202,* 218, *232,* 249, *264,* 249, *265,* 364, 372, *373,* 387, *397,* 387, 395, 396, *398,* 406, 408, *416,* **23, 387-398**
Rhoades, M.V., 364, *372*
Robillard, J.E., 61, *63*
Robinson, J.S., 111, *122,* 113, *123*
Robinson, P., 237, *245*
Roby, T.B., 42, *46,* 171, *172,* 357, *362*
Rodin, J., 291, *294,* 297, 299, 301, 304, 305, *307,* 308, *308,* **22, 280, 281, 295-308**
Rosen, S., 346, *349*
Rosenthal-Zifron, I.A., 174, *188*
Roswell, V., 310, *325,* 310, 313, *326*
Rothschild, E.J., 160, *160*
Rozin, P., 19, *20,* 43, 45, *46,* 218, 225, *232,* 336, *344*
Rubal, L., 345, *350*
Rusiniak, K.W., 19, *20*
Russell, A.L., 345, 346, *349*
Rytand, D.A., 295, *307*
Rzoska, J., 218, *232*

Safford, H.R., 42, *46*
Sagerson, R.N., 61, *64*
Saggers, B.A., 156, *158*
Salan, L., 120, *123*
Salapatek, P.H., 209, *212*
Salisbury, D.M., 107, *107,* 113, 120, *123,* **111-123**
Sameroff, A.J., 146, 147, *158*
Sampson, J., 88, *98*
Samson, H.H., 376, 377, *385,* 377, 378, 381, *386*
Samuelson, G., 241, *245*
Sato, M., 26, *39*
Schachter, S., 297, 304, 305, *307,* 314, *326*
Schaller, G.B., 331, *344*
Schiffman, H., 35, *39*
Schlosberg, H., 194, *203*
Schulz, M.D., 58, *62*
Schulz, T.W., 358, *362*
Schuster, C.R., 384, *386*
Sclafani, A., 295, *307,* 309, *326,* 341, *344*
Scott, C.R., 61, *64*
Scott, E.M., 243, *345,* 249, *265*
Scott, J.W., 20, *21*
Segal, A., 184, *187*
Semb, G., 146, *158*
Semer, J.M., 160, *160*
Senning, R.S., 348, *349*
Seward, J.P., 234, *234*
Sharma, K.N., 101, 102, 106, *107,* 285, 292, *294,* 337, *344*
Sharpless, S., 209, *212*
Shaw, J.H., 347, *350*
Sheffield, F.D., 42, *46,* 171, *172,* 357, *362*
Sheldon, W.H., 274, *279*
Shelley, H.J., 88, *98*
Sherrington, C.S., 65, *65,* 193, *202*
Sherry, D.F., 51, *63,* 227, *231*
Shirley, M.M., 130, *140*
Shuford, E.H., 22, *22,* 42, *47,* 357, *362*
Silverstone, J.T., 248, *248*
Shinn, M.W., 126, 127, *140*
Simms, M.E., 348, *349,* 351
Simone, M., 323, *326*
Singer, J.E., 305, *307*
Singh, D., 310, *326*
Sisson, S., 87, *98*
Skoloda, T.E., 384, *386*
Slater, J.S., 61, *63*
Slochower, J., 305, *307*
Smallman, R.L., 73, *83*
Smith, D.C., 298, *306*
Smith, D.V., 317, *325*
Smith, F.G., 61, *63*
Smith, G.K., **70-83**
Smith, M.P., 357, 359, *362*
Smith, R.D., 16, *21*
Smutz, E.R., **99-107**
Snyder, H.L., 357, *360*
Snell, T.C., 13, *21,* 26, 27, *39*
Sonohara, T., 130, *140*
Sorenson, C.A., 16, *21*
Spencer, M.M., 249, *263*
Spiegel, T.A., 306, *307,* 315, *326*
Springer, D., 309, *326*
Squibb, R.L., 333, 336, 339, *343,* 338, *344*
Stanley, W.C., **64, 232**
Stare, F.J., 331, *344*
Stein, M., 148, *158*
Steiner, J., 107, *107,* 131, *140,* 174, 184, *187,* 178, 179, 184, *188,* **22, 23, 141, 158, 159, 173-187, 188, 189, 203**

Steininger, von F., 218, *232,* 232
Štellar, E., 166, 167, 170, *172,* 191, *202,* 295, 297, 306, *306,* 307, 309, 310, 312, 315, *325,* 331, *343,* 333, *344,* **327, 328, 350, 363-373**
Stephan, R.M., 345, *349*
Stern, I.B., 52, *62*
Stern, J.S., 313, *326*
Stevens, S.S., 274, *279,* 282, 284, 285, 288, *294*
Stewart, C.N., 43, 46, *46,*
Stewart, R.A.. 235, *245*
Stiles, H.M., 347, *349*
Stirnimann, F., 130, *140*
Stockton, M.D., 51, *64*
Storey, A.T., 115, *123,* 193, *202*
Strom, L., 7, 9, *20*
Strouthers, A., 357, *362*
Stunkard, A.J., 166, 170, *172,* 248, *248,* 315, *326,* 369, *372*
Sun, K.H., 126, 128, *140*
Sunday, S., 339, *339*
Suzuki, S., 249, *263*
Swaminathan, M.C., 238, 239, *245*
Symington, L.F., 101, *107,* 269, *279*
Swarup, S., 238, *245*
Sweet, C., 249, *265*
Swenader, Lanke, L., 346, *348,* 351

Takagami, H., 252, 260, *264*
Takaheshi, M., 249, 263, *264,* 249, 260, *265*
Takeuchi, M., 345, *350*
Tallon, S., 51, *63*
Tapper, D.N., 42-45, *46,* 47, 48
Tateda, H., 26, *39*
Teitelbaum, P., 16, *20,* 93, *97,* 295, 297, *307,* 331, *344,*
Telle, H.J., 218, *232*
Teng, C.C., 61, *64*
Thach, B.T., 131, *140,* 205, 209, 211, *212,*
Thayer, K., 61, *63*
Thompson, D., **328, 329**
Thompson, M.M., 42, *46*
Thompson, T., 384, *386*
Thomson, L.A., 347, *349*
Tindale, N.B., 5, *21*
Torrey, T.W., 70, 71, *84,* 85, *85,*
Toverud, G., 345, *350*
Trexler, P.C., 347, *349*
Tso, W., 187, *187*
Tsuda, R., 254, 260, *264*
Tucker, W.B., 274, *279*
Tuntland, P., 42, *46,* 357, *361*
Turner, R., 67, *67,* 91, *97,* 148, 155, *157,* 161, 162, *172*
Turner, R.E., 9, *20,* 51, *63,* 143, *145,* 298, *306*

U.S. Department of Agriculture, 241, *246*

Valenstein, E.S., 42, 43, 46, *46,* 48, *48*
Vander-popuhere, J.M., 93, *98*
van Horne, W., 285, *294*
van Itallie, T.B., 295, *306,* 314, *325*
Veal, W.L., 375, 380, *386*
Vitale, J.J., 331, *344*
Vivian, V.M., 240, *244*
von Ebner, V., 71, *83*
von Vintchgau, M., 70, *84*

Wagner, M.G., 347, *349,* 359, *362*
Wahlin, C.S., 388, *397*
Walicke, B.C., 306, *307*
Walker, J.W., 320, *326*
Wallance, H.D., 93, *98*
Waller, R.F., 274, *279*
Wampler, R.S., 310, *326*
Wang, S.C., 190, *202*
Ward, A., 411, *416*
Warren, R.M., 7, *21*
Warren, R.P., 51, *64,* 233, *233*
Waterman, D., 101, *107,* 269, *279*
Weaver, M.E., 88, *98*
Webber, R.L., **68**
Weber, M.L., 42, *46*
Wehrly, T., 285, *294*
Weiffenbach, J.M., 107, *107,* 131, *140,* 205, 207, 209, 211, *212,* **XI, 41, 65, 66, 69, 205-212, 213**
Weiland, W., 166, 170, *172*
Weiner, D., 174, *188*
Weistreich, V., 61, *62*
Welch, N.N., 88, *97*
Wellin, E., 237, *246*
Wells, H., 42, *46*
Werther, P., 184, *187*
West, E.A., 235, 240, *245*
Westerling, J., 269, *279,* 289, 290, *294,* 318, *326*
Whiteside, B., 70, *84*
Whitney, G., 51, *64*
Wickes, I.G., 236, *246*
Widdas, W.F., 61, *62*
Widdowson, E.M., 337, *344*
Wiehl, D.G., 345, *350*
Wieland, W.F., 369, *372*
Wiesel, T.N., 52, 61, *63*
Willett, N.P., 347, *350*
Wilson, M.R., 89, *98*
Windle, W.F., 58, *62*
Winger, G., 376, 377, *385,* 377, *386*
Wohlzogen, F.X., 61, *62*
Wolff, P.H., 120, *123,* 133, *140,* 146, *157,* 146, 147, 148, 155, 156, *158*
Wolstenholme, G.E.W., 348, *350*
Wood, M.W., 388, *397*
Woodburn, J., 242, *245*
Woodworth, R.S., 194, *203*
Wooley, S.C., 306, *307,* 315, *326*
Wright-St. Clair, R.E., 243, *246*
Wyatt, D.G., 58, *62*

Yamashita, S., 26, *39*
Yensen, R., 319, *327*
Yin, T.H., 22, *22*
York, D.A., 295, *306*
Young, J.Z., 202, *203*

Young, P.T., 42, *47*, 106, *107*, 249, *265*, 299, *307*, 357, *362*, 372, *373*, 402, 403, 404, 405, 406, 407, 409, 410, 412, *416*, *417*, **399-417**
Yudkin, J., 241, *246*

Zalewski, A.A., 70, *84*
Zebley, S., 166, 170, *172*, 369, *372*
Zinner, D.D., 347, *348*
Zipkin, I., 347, *349*
Zotterman, Y., 9, *19*, 7, 9, *20*, 123, *123*
Zucker, L.M., 313, *325*
Zuckerman, H., 174, *187*
Zukerman, H., 61, *62*

Subject Index

Acid: role of, in caries, 346
Across fiber response correlations, 34, 35
Activity, 281, 339-341
Adaptation: to stimulus for neonate's tongue movement response, 207
Adaptive choice: in neonatal pig, 92; inappropriateness of, in altered environment, 341; time scale of, in relation to salt homeostasis, 414
Adipose tissue: composition of, in obesity, per cell size and number, 313; composition of, in relation to diet and weight status, 280
Adult, human: facial responses in, 184; preferences of, relative to adolescents, 168, 169; preferences of, relative to newborns, 166; sensitivity of, relative to newborns, 130; odor preferences of, in comparison with young children, 146
Adult, rat: effects on weanling feeding, 222, 223; effects on adult feeding, 233
Affect: neurological representation of, in the activity required to inhibit acceptance and rejection reflexes, 191; positive and negative, 339. *See also* Hedonic effects of stimulation
Age: at first introduction of sugar in rat, 258; at first introduction of diet items in children, 254, 255; effects of increments in, on intake of sweets, 261; in relation to protein requirements in rat, 337
Age comparisons: adult with newborn pig, 92; infant and adult with newborn human, 165, 166; lamb with fetus and adult sheep, 55
Airway defense: failure of, in human infants during ingestion of saline, 119; and aversive reactions to feeding, 138
Alimentary canal: comparison of, in man and pig, 88
Alliesthesia, 292
Amino acids: receptors for, 14, 23
Amniotic fluid: response of chorda tympani to, in fetal sheep, 56, 57; swallowing of, 58-60; as a taste environment, 61; failure of, to elicit either apnoea or swallowing in newborn lamb, 114, 115
Anterior-posterior tongue areas: 6, 7, 8, 16, 194
Anthropomorphism: of investigators studying human neonatal taste, 293
Antibiotics: in the control of caries, 347, 350
Aphagia: produced by chemical lesions, 16
Apnoea, 113, 117
Arytenoid process: taste buds on, in fetal sheep, 115
Assimilability, limits of: and sugar ingestion in rat, 391, 392 ff, 396
Averaging: effects of, on shape of developmental curves, 102-104; masking of relationships by, 287
Aversion: to saccharin, 90; to sucrose, 317, 319, 323, 324, 328; to sodium chloride, 317; to feeding, 138; oral and post-ingestive factors in, 364. *See also* Taste aversion

Blindness: effects of, on tendency of rat pups to eat their first meal in the presence of a feeding adult, 224
Balance of protein and carbohydrate in diet: defense of, relative to defense of caloric intake, 337, 342; changes in, with age, 338; effects upon, of cold stress, activity, deprivation and food availability, 339-342
Balanced nutrition: in humming birds, 22
Between meal snacks: in relation to low caries level of institutionalized children, 345, 351
Beverages: preferences for, among blacks, 273. *See also* Drinks, soft
Bias: in self report of food preferences, 280
Birds: sweet preferences in, 3, 4; differences among, 22
Bitter: rejection of, by human newborn, 100, 101, 106; as an aid in weaning human infants, 247
Blacks: sweet and saltiness preferences of, 168; beverage preferences of, 273; food preferences of, 270-274
Blood pressure: measurement of, in newborn lambs, 112
Body fat: as a function of early access to sugar or chocolate in rat, 261, 262; composition of, per cell size and cell number in obesity, 313; insufficient quantity of, for thermal insulation of neonatal pig, 89
Body length: retarded development of, in rat due to mismatch of pup's age with post-partum age of foster mother, 80
Body movement: recording of, in newborn human taste tests, 128, 129
Body size: comparability of, for man and pig, 86
Body weight: relation of, to sensory aspects of sweet taste, 299; relation of, to responsivity to the taste of food, 295, 296; as a function of early diet in rats, 261, 262; failure of long-term changes (reductions) in, by obese adolescents to alter attractiveness of sweetened solutions, 322; failure to maintain, under time restricted concurrent availability of sucrose and food, 341, 384; of rat pups reared by mothers mismatched for post-partum age, 80; complexity of relation with food preferences, 279; regulation of, by rats offered a highly palatable supplement to their normal diet, 332
Body weight categories: variation in definition of, and altered findings in neonate human, 298; variation in definition of, and altered findings in human adults, 277, 278; definition of, in terms of set point, 301; definition of, in terms of weight stability, 302; somatotype ponderal index, 274. *See also* Metropolitan Life Tables
Bottle feeding: rise of, in the U.S. and elsewhere, 240
Brain mechanisms: cortical and subcortical, 18, 19, 415

Brain stem: and the organization of ingestive reflexes, 18
Breast feeding: decline in, 240
Breast milk: rejection of, by human neonate, 101, 106; declining use of, and increasing use of sweeteners in infant feeding, 239, 240; regular breathing pattern during feeding of, 220
Breathing: regularity of, during burst-pause sucking, 120, 133; differential disruption of, by various ingested fluids, 120, 121, 133-135
Brief exposure preference tester, 401
Burst-pause pattern of sucking, 120, 121, 133, 147, 210

Cabanac effect, 296, 297, 301, 320, 370, 371, 413, 414
Calories: sources of, in diets of high and low income children, 241; in commercial baby food, 235; from sweet solutions, 356
Caloric intake: role of, in determining total dietary intake in rat, 393, 396; of rats on sweet supplemented diets, 261, 262; decrease in, with selection of sodium saccharin, 379; defense of, in relation to defense of protein carbohydrate ratio, 337
Cardiovascular responses: to upper airway perfusion in newborn lamb, 113; differential elicitation of, by artificial and breast milk, in human neonate, 121; to sweet fluids in newborn humans, 127, 131, 132
Caries, 241, 249-251, 254, 255, 345, 374, 400; absence of, in pig, 88; among Hadza, 242; and diet, 250; and meal pattern in rat, 348
Cat, decerebrate: feeding behavior of, 18, 19, 193, 194
Category rating scales: in measurement of taste response in neonatal rat, 101; effects of increased number of categories in, 104
Category scales: definition, 284; cautions in use of, 284
Catfish: distribution of taste buds in, 17; feeding behavior of, 17, 192
Central nervous system pathways: in rat, 14, 15, 16, 17, 23; in catfish, 17; in man, 193, 194; in phylogenetic perspective, 17-19
Central representation: hierarchical organization of, 19; of a behavioral distinction, 201
Chemical cues: in control of feeding behavior of rat pups, 222, 223, 229, 230
Chemotaxis, 187
Chocolate: effects of, on health and development of young rats, 260, 261, 262; most preferred "sweet" among children of both sexes, 253, 254; exposure of Japanese children to, 255
Choice behavior: objection to, as an indicator of preference, 290
Chorda tympani: recordings from, in humans and their role in inferring sweet taste in animals, 7; response differences among single fibers of, 11-14, 27-41; whole nerve responses from, in rat, hamster, and squirrel monkey, 25, 26; single fiber recordings from, in hamster and squirrel monkey, 27-39; single fiber recordings from, in rat in comparison with hamster, 40, 41; recordings from, in fetal, newborn, and adult sheep, 53-57; comparisons of response from, with response from glossopharyngeal, 7, 8; role of, in reflex mechanisms of feeding, 192-195
Citric acid: human newborn's response to, 163, 174
Coarse structure: of sucking behavior in contrast to fine structure, 147
Cognition: in relation to affect, 415, 416
Cold stress: and increased intake of carbohydrate in rat, 339
Colostrum: human, prohibitions surrounding, 236, 237; role of, in protecting the health of neonatal pig, 89
Communicational value: of facial expressions, 175, 179, 189
Comparisions among sensory systems: taste with olfaction in newborns, 144; taste with vision in regard to effects of experience, 61
Comparisons among species, age groups, weight groups, races, and measures. *See* individual entries
Comparisons among cultures: *See* Cross-cultural comparisons
Complex stimuli: simultaneous elicitation of several taste qualities, 14; in taste testing of human neonates, 163; neglect of, by taste investigators, 269; in the study of preference and obesity, 298 ff, 317, 322
Computer assisted analysis of sucking, 132 ff, 143 ff
Concentration: of sugar solutions, effects on amplitude of elicited anterior tongue movements, 196, 197; specificity of learning to, 48, 359, 360. *See also* Seriation
Conditional enhancement, 380, 381
Conditioned taste aversion, 19, 44 ff
Congenital, 161. *See also* Newborn
Consummatory behavior, 11
Consumption (Intake): high correlation of, with preference ratings for sucrose among normal weight, moderately obese, and severely obese subjects, 318
Continuous sucking: elicitation of, during feeding of artificial milk to human newborns, 120
Contrast effects, negative: in human newborn, 139; failure of, to reach statistical significance, 144
Cortex: cortical gustatory area, 15, 16; absence of, and differential gestural response to taste stimuli, 175
Craniofacial malformations: reconstructive surgery for, and the role of taste and smell testing, 184, 185
Craving, physiological or biochemical: dominant role of, in the theory of addiction, 383, 384
Cross-cultural comparisons: of infant feeding practices, 235-248; of the schedule on which various foods are introduced, 254, 256, 257
Cross fiber pattern: theory of taste quality coding, 14
Cross fostering: effects of, on development of lingual papillae in rat, 77, 78; failure of, to measurably disturb maturation, in rat, 79–81
Cultural differences and similarities: in regard to exposure to sweets, 254, 256, 257; in infant feeding practices, 235-248
Cyanosis during feeding: in lambs, 117, 123; in humans, 121, 122, 123

Dental Health. *See* Caries
Dentition: in rat, 86; in pig, 87, 88
Dependence, physical: upon ethanol, establishment of, per schedule induced polydipsia, 375-377; failure of, to explain reinforcing properties of drugs, 384, 385

Demographic information: from respondents to food preference questionnaire, 270; in the analysis of obesity, 241, 248; and diet surveys, 249-251
Desserts: more highly preferred by underweight than overweight individuals, 274, 275; poor representation of, among the most preferred foods, 270; preference for, among Blacks, 273
Desserts, chocolate: possible role of, in differential exposure to sweet for Whites and Blacks, 274
Dextrose: energy value of, in relation to activity and survival in rats, 295; preference for, relative to ethanol, 378, 379. *See also* Glucose
Diabetes, 45, 241
Diet: similarity of, in pig and man, 87; as a possible etiological factor in dental caries, 250; surveys of, 241, 243, 249-251; locating of, as a problem for weanling animals, 217 ff; locating and swallowing of, dissociated in ablation studies, 18, 19
Diet, basal: intake of, when sugar or chocolate supplement is available, 261, 262; neglect of, under time limited exposure to diet plus sucrose, 341, 384
Dietary experience: failure of differences in, to effect human preferences, 166; with sweets not a necessary preconditon for a preference for sweets, 243; with sour in relation to measured sour preference, 247
Dietary sweet: absence of, among certain tribes, 242
Diet surveys, 241, 243, 249-251
Dilute water effect, 368
Dilution experiments: alternative conceptualization of 24-hour intake preference studies, 333, 334, 335
Dissociation: of locating and swallowing food, 18, 19
Distilled water. *See* Water
Domestic rats: difference of, from wild rats in "neophobia," 225
Drinks, soft: in childhood feeding, 240, 241, 243; preference for, in adults, 270; preference for, among Blacks, 273; hedonic ratings of, modified by long term weight reduction in obese adolescents, 232; preference for in piglet, 92

Early experience: *in utero,* 51 ff; pre versus post natal 62; effects of, on the development of taste papillae in rat, 74 ff; with test fluids nonessential role of, in the development of sweet acceptance and quinine rejection in rat, 102; possible effect upon exploration, 139; of sweets in pre-lacteal feeding of human infant, 236-238; with sweet taste, 241; with "sweets" not a pre-condition for later acceptance or preference, 243; cultural similarities and differences in, 254-257; timing of, fails to produce effect on later preference in rats, 260
Eating: temporal patterns of, 64, 65, 348
EEG: in defining state in newborn lambs, 111 ff
Energy requirements: increases in, with low temperature, food deprivation and access to exercise, 339 ff
Entrees: race differences in preference for, 272; weight related differences in preference for, 276
Environmental factors: in instituting, maintaining or suppressing ethanol abuse, 383, 384
Eskimos, 243, 345
Ethanol abuse: establishment of, in animals through schedule induced polydipsia, 375 ff; environmental determinants of, in man, 383, 384
Evaluative reaction, 415
Experience: denial of the necessity for, in the human neonates' development of differential facial responses to taste, 175; role of, in modification of innate response to sensory stimuli, 359
Epiglottis: taste buds on, in fetal lamb, 115
Exploratory behavior: possible effect of early experience upon, 139
Expressed breast milk: force fed to rat pups prior to lithium chloride treatment, 227
Experimental obesity: *See* VMH lesions
External cues: responsivity to, as a causal factor in obesity challenged, 314, 315, 317; manipulation of, per addition of color, effects on magnitude estimations of sweetness in obese and normal weight subjects, 317

Facial responses: of rejection in human newborns, 126; in relation to sucking, 129; increased display of, with age, 218, 219; resemblance of, between newborn and adult, 126, 217; elicitation of, by taste, 174-178; elicitation of, by food related odors, 178, 179; display of, prior to feeding experiences, 175; innate, 186; as indications of acceptance and rejection, 99-100; in rat and hamster, 101-107
Fat: in relation to other diet elements, 23, 334
Feeding: respiratory and cardiovascular components of, not to be neglected, 122
Feeding behavior: rejection of, for measurement of taste sensitivity, 205, 211; rejection of, for the measurement of taste preference, 203, 204
Feeding reflexes: disruption of, by CNS manipulation, 16, 17, 18, 297
Feeding rites: neonatal, 236
Fetal taste: developmental morphology of, 52; electrophysiology of, 52 ff; and fetal swallowing, 58-62
Fetal rats: fungiform papillae on the tongue of, 71, 85
First meal of solid food: ingestion of, in the presence of feeding adult rat, 223, 224
Fine structure: of sucking behavior in contrast to coarse structure, 147
Fish and fish products: Black-White differences in preference for, 272
Flavor cues: learning of, 411, 412
Fluoride, 348
Fluid: effects of response contingent delivery of, on fine structure of sucking, 147
Fluid touch, 211
Folk practices of U.S.: endorsement of sugar use by, 240; in comparison with Japanese practices, 254, 257
Food deprivation: stimulation of eating by, in neonate pigs suckling from sows, 95; and the dynamic phase of VMH lesion induced obesity, 310 ff; and taste preferences, 319, 320, 322; long term, and taste, 320, 322; adaptation to, with and without sucrose availability, 341, 342; and palatability, 357; and schedule induced polydipsia, 374, 375

Food habits: role of, in stabilizing intake, 411
Food preferences of Blacks: assessment of, by questionnaire, 271 ff
Food preferences of Southerners: in the analyses of Black food preferences, 271
Frequency of exposure to sweets: among Japanese children, 254, 258
Fructose: response to, in relation to other sugars, 8 ff; intolerance of, and low levels of caries, 345, 350; preference-aversion curves for, in rat, 364; intake preference thresholds for, in rat, 389, 390; maximal intake of, in rat relative to other sugars, 394, 395; energy value of, in relation to activity and survival in rat, 395
Fruit drinks: race differences in preference for, 273. *See also* Drinks, soft

Galactose: intake preference thresholds for, in rat, 389, 390; maximal intake of, relative to other sugars in rat, 394-395
Gastric capacity: possible limitation on food intake of piglets, 96
Gastric distension, 371
Gastric loading: to manipulate water imbalance and clarify differences between hunger and thirst, 357
Genetic factors: role of, in sweet preference, 51; role of, in preference, 161; failure to find effect of, 168
Glossopharyngeal: sensitivity to quinine, 7, 8
Glucose: response to, in relation to other sugars, 8 ff; regulation of, similarities between pig and man, 88; preference for, in fasted neonatal pigs, 91; effect of, on sucking in newborn human, 143; response to, in human newborn, 197; as example sweet tastant in study of human neonate, 207-211; feeding of, in U.S. hospitals, 236; intensity and hedonic tone category scale judgements of, by the same individuals, 285; other stimulus properties of, 356; preference-aversion curves for, from rat one hour intakes, 363; human preference-aversion function for, by ten minute intake, 369; intake preference thresholds for, in rats, 389, 390; maximal intake relative to other sugars in rat, 394, 395
Gymnemic acid: specific blocking of sweet, 7

Habituation: in neonatal testing, 209
Habits of dietary selection, 400
Hamster: chorda tympani response profiles of, 27
Heart rate: recordings of, in newborn lambs, 112; relation of, to rate of sucking during sucking burst, 134; increases in, as a function of sweetness, 137; difference in, between resting, no fluid sucking and sucrose sucking conditions, 137
Hédonic dimension of stimulation: and the communicational aspect of facial expressions, 186; role of, in the organization of behavior and generation of preferences, 400
Hedonic effects: of stimulation, 399, 404, 414; independence of, from learning, 404; separation of, from sensory effects, 414
Hedonic equivalence function, 405
Hedonic evaluation: possible unipolar nature of, in newborn human, 144; independence of newborn human's lateral tongue movement from, 210
Hedonic judgements: of food items, 270; comparison of, with intensity judgements, 285, 370, 371; as a response to intrinsic properties of a stimulus, 355; comparison of, with intake measures in human adults, 369
Hedonic monitor, 173, 182, 184, 187
Hedonic responses: role of, in control of short term intake, 164; relative strength of, to taste and odor in human neonate, 144
Hedonically negative oral stimuli: human neonatal response to, 99-101, 126-131, 162, 163, 175-178, 197; age related changes in human response to, 67; evidence for responses to, in relation to biopolar hedonic scales, 144
Hedonically negative odors: human neonatal response to, 179 ff
Honey: in pre-lacteal feeding, 236, 238; in infant diets, 242
Honey ants: role of, in the diet of hunter-gatherers, 5
Honey glider, 22
Homeostases, 414
Homeostatic need: effects of, on preference, 410, 411
Human milk (expressed breast milk): ingestion of, by human infants by regular burst-pause sucking does not disrupt breathing, 120; consistent rejection of, by human neonate, 101
Human newborn: 99, 100, 101, 106; bitter rejection, 100, 101; sweet acceptance, 100; gestural or facial responses to taste solutions in the mouth, 99, 100
Hunger: effects of, on preference, 16; effects of, on palatability, 357; reports of, differentially correlated with gastric motility in normal weight and obese subjects, 315
Hunter-gatherers, 4, 5, 235, 242
Hyperphagia: reflects loss of rostral control over intake and rejection reflexes, 191; absence of weight gain in VMH rats on unpalatable diets, 309
Hyperresponsiveness to taste: in rats with VMH lesions, 309
Hypertonic sweet solutions, aversion to: oral and postingestive factors in, 364 ff

Indians: North American, 243
Indicator behaviors: choice of, 203, 204, 290, 293, 333-335, 355, 356, 404, 409
Individual differences: in fetal swallowing, 59; among human adults, 167, 170, 171; and the study of unusual individuals, 263; possible masking of, in grouped data, 102, 103, 287; in the preferences of rats, 102-104, 402
Infant diets: exclusion of certain foods from, 246
Infant feeding practices: ready modification of, 244
Infant food, commercial: addition of sugar to, 240
Ingestion: inability of newborn to inhibit, 67; at the nest site and subsequent food preference at weaning, 223; objections to use of, as an index of preference, 203, 204, 290, 293, 333-335, 355, 356, 404, 409

Inhibition of reflexes: failure of, in newborn, 67; neuronal activity implied by, 201
Innate: responses of neonates, 106, 174; neural structures for hedonic response, 399
Instinctive reactions, 415
Instinctive rejection: by odor in human newborn, 182
Insulin: in producing glucoprivic eating, 93; age dependent effect of, in pig, 94, 95; in separation of hunger from thirst, 357
Intake as an index of preference: in children, 319; in human adults, 314 ff, 369; in rats, 310 ff, 374 ff, 387 ff; in human newborns, 162 ff; in pigs per weight gain, 94; challenges to, 203, 204, 290, 293, 333-335, 355, 356, 404, 409, 410; indirectly measured per consummatory behavior, 203
Intake, regulation of: by selective facilitation or inhibition of reflexes, 190, 191
Intensity judgements of: compared with hedonic judgements when sweet test samples are repeatedly ingested, 370, 371
Interpretation: in the generation of pleasantness functions, 290, 291
Interval scaling, 284
Intolerance: of fructose, 345, 350; of sucrose, 243
Intrauterine behavior: monitoring of and attempts at manipulation of, 58-61; relevance of, for facial expression in newborn, 186
Isohedons, 405

Labelled line: theory of quality coding, 14
Lambs, newborn: similarity of, to fetus and to adult in taste responding, 55; swallowing in, 112-114; elicitation of apnoea in, per water, 113, 114, 116; superior laryngeal nerve response in, 115; chemosensitive receptor of, 114, 115
Lactose: stimulus effectiveness relative to other sugars, 10, 387 ff; rejection of, by rats, 389, 390, 394, 395; energy value of in relation to activity and survival in rat, 395
Language: role of, in distinguishing intensity from hedonic determiners of choice, 290
Learned safety: associated with glucose or glycine generalization of, to saccharin, 43, 44
Learning: in the newborn human, 139; of food preferences rather than aversions by weanling rat, 221, 222; modulation of palatability response by, 359, 360; and the stabilizing role of food habits, 411, 412
Length: retarded development of, due to mismatch of rat pup's age with postpartum age of foster mother, 80
Length of sucking burst: relation of, to sweetness of ingested fluid, 133, 134
Licking rate: in rat, 159, 412, 413; failure of, to discriminate among sugars showing seriation per bar pressing, 11; differential patterns of, for sugar and saccharin, 159
Lithium chloride: introduction into food, 218 ff; injection of, 227
Locating of food: in hunter-gatherer cultures, 4, 5; disruption of, by ablation of facial lobe in catfish, 18; hazards of, for weanling rat, 217; differences in task of omnivore, herbivore and carnivore, 330
Magnitude estimation: a method for ratio scaling, 288
Maltose: intake preference thresholds for, in rat, 389, 390; maximal intake of, in rat relative to other sugars, 394, 395; energy value in relation to activity and survival in rat, 395
Marketing and distribution practices: effects of, on diet, 243
Maturation of taste buds: differences in, and developmental differences in acceptance and rejection 102
Maturational retardation: production of, in rat by mismatch of litter age and maternal postpartum age, 79-81
Meal patterns: in the generation of caries in rat, 348
Measures of sucking, 132
Mechano-stimulation: in the elicitation of sucking, 68, 69; response to, by the human neonate's tongue, 205; in association with drinking, 356
Metabolic state: failure of short-term changes in, to effect preference for sucrose in normal and obese children and adolescents, 322
Metropolitan Life Tables: use of, in defining over and underweight and degrees of obesity, 277, 295, 327, 328
Microflora: in caries development, 347, 348
Microorganisms: oral, essential role of, in the generation of caries, 347, 350; intestinal, defense against, in piglets by sow's milk, 89, 90
Micro structure: of sucking, 147
Milk: lesser preference for, by Blacks than Whites, 274
Milk, artificial: preference for, over glucose solution by fasted piglets, 92; disruption of breathing during ingestion of, 120-122; low preference for, relative to sucrose solution in human neonate, 163, 164
Milk, cow's: mild acceptance of, by human neonate, 100, 101, 106; elicitation of apnoea and swallowing by, from the airway of newborn lamb, 114, 115; failure of, to elicit differential response in human neonate, 128
Milk, mother's: rejection of, by human neonate, 101; failure of, to elicit differential response in newborn human, 128; as source of flavor cues to maternal diet, 225-229
Milk, sweetened, 162
Mimetic reaction: of newborn humans to taste stimuli, 126-130, 173 ff; of newborn humans to food odors, 178-182; in anencephalic infants, 182-184; social control of, in adults, 184
Motivation to obtain sweets: assessment of, in humans, 299
Mouth sensory effects: in the production of satiation, 358

Neophobia: and the prenatal chemical environment, 61, 62; role of, in maintaining initial diet choices by weanling wild rats, 225
Neural development: of the taste system *in utero* per electrophysiology, 52-57; of cross-fostered rat pups, 81
Newborn human: comparison with adults, 129, 166; regurgitation in, 67
Newborn human, measurement of taste sense in: historical summary, 125-131; per facial reaction, 99-101, 126-130, 173-178, 182, 183; per intake, 129, 161-166;

per perameters of sucking, 132-137, 143, 144, 146-157, 195-198; per cardiac response, 131-139; per lateral tongue movements, 205-212
Newborn pig: glucose and sucrose preference in, 91, 92, 97. *See also* Pig
Newborn sheep. *See* Lambs
Ninth cranial nerve, 6, 14; activity combined with seventh nerve activity to predict choice behavior, 8, 198, 199
Nipple: for presenting stimuli and recording sucking, 118, 132, 143, 148; possible difference between free and restricted flow varieties, 141; for recording tongue pressures and delivering fluids, 195, 196
Nominal scaling, 282, 283
Novelty: in acquisition of taste aversions, 61, 62; avoidance of, by weanling rats, 225; failure of, to account for postadaptation responding of human newborn in lateral tongue movement taste testing, 209
Number of responses per burst: day to day stability of, in the sucking of human neonate, 137
Nutrients: changing need for, in the control of food intake in growing organisms, 262, 337
Nutrition: lack of information concerning effects of early differences in, on subsequent taste preference, 324
Nutritional value: strong relation of, to preference, 408, 409

Obesity, 241; in relation to use of sugar or sweeteners, 248; in relation to socio-economic status, 248; and selective overindulgence, 280; correlation of, with monotonic pleasantness x intensity functions for sweet and salty, 291, 292; lack of established causal relation with taste responsiveness, 298; differential effects of, on preference for sucrose and aversion to NaCl, 317; degree of, and sucrose aversion, 317; degree of, in conjunction with Metropolitan Life Insurance Tables, 277, 295, 327, 328
Oral satiety, 358
Ordinal scaling, 283
Odors: intensity of hedonic reactions to, 144; discriminative response to, per facial expressions in newborn human, 179-185; response to, in anencephalic neonate, 182, 184, 185; in relation to feeding reflexes, 203; as cues in the transmission of feeding preferences, 232, 233
Olfactory stimuli. *See* Odors
Olfactory system: interactions of, with gustatory system, 21
Olfactory bulb: developmental schedule of, 189
Omnivore: pig, as a laboratory model for, 86; dietary problems of, fundamental differences from those of carnivore or herbivore, 330, 331, 343
Ontogenetic comparison: of protein selection in rats, 23. *See also* Age comparison; Adult, human
Operant conditioning: in infant human, 138
Operant response: in test of relative effectiveness of sugar for squirrel monkey, 10; performance of, by pigs to obtain sucrose, 90; in manipulating availability of diet, 342; and schedule induced polydipsia, 374 ff
Oral factors: in satiety, 258, 259; in human preference and aversion, 370; osmotic, 365
Oral hygiene: unimportance of, in caries attack on humans eating high starch diet, 346
Osmolarity: role of, in ingestive preference, 21, 22
Osmotic factors, oral: in contrast to taste factors, 365
Osmotic pressure: as a stimulus property of solutions used as stimulus for sweetness, 356
Overindulgence: in sweets or carbohydrates, absence of adequate observational base for linkage of, to human obesity, 323; massive, in the definition of abuse, 374; selective, in obesity, 280
Overnutrition: infantile, in the production of obesity, 313, 314
Overingestion: of sweet fluids in the time limited two bottle testing of squirrel monkey, 9. *See also* Overindulgence
Overweight humans: food preferences of, per questionnaire, 274-278; willingness of, to work for preferred and non-preferred rewards relative to normal weight subjects, 300; similar differences of, from both normal and obese in intake, hedonic judgements and effects of increased effort, 303-305. *See also* Obesity

Palatability: and sweetness, 355 ff; as the hedonic response to food, 400 ff; and the relative activity of ninth and seventh nerves, 8, 198, 199; and reflexes of acceptance and rejection, 198; reflection of, by drinking only in the initial minutes of presentation, 357, 358; change in, as the mechanism for gastric inhibition of intake, 359; reduction of, for the protein component of a diet and parallel reductions in both protein and carbohydrate intake, 336, 337; modulation of, by learning, 359, 360
Pancreas, 22, 23
Papillae: location and morphology of, 5, 6, 7; schedule of development for, in sheep, 52-55; and taste pores, 52, 85; fungiform and filiform, differential staining by methylene blue, 71, 72; fungiform, failure to discern on tongue of fetal rats, 72, 85; fungiform developmental change in number in rat, 72, 73, 74; effects of environmental manipulations on the calendar for developmental change in number of, 74-79
Pauses during sucking: decreases in, with increases in sweetness of ingested fluid, 134; effects of NaCl upon, 130, 210. *See also* Burst—pause pattern of sucking
Peripheral sensory mechanisms: for taste reception on the tongue of the neonate human, 210, 211. *See also* Papillae
Pharmacological action of addictive agents, 383
Phylogenetic relations, 17 ff. *See also* Species differences and entries for specific species
Physical growth: in rats, 79-82, 254, 262
Pig: similarities of, to man, 86, 87, 88; similarity to low birth weight human based on neonatal lack of fat and glycogen stores, 88; special procedures for laboratory study and maintenance of, 89; resemblance of insulin sensitivity of, to that of human and dog but not rat, 93; neonatal glucose and sucrose preference of, 91, 92, 97;

social facilitation of sucking in, 95; development of glucoprivic eating in, 93-97; effects of food deprivation on, 95-97
Pleasantness: contrast of, with other possible controls on ingestive behavior, 293, 294; quantification of, separately from sensory quality or intensity, 355
Poison avoidance, 218
Polydipsia, 374, 375
Polysaccharides, extracellular: role in caries, 347
Pons, 24
Pore, taste: in photomicrographs, 5, 6; in relation to taste papillae and taste buds in sheep and in fetal human, 52-55; as an index of number of taste buds, 85
Posterior tongue: responses of, to quinine hydrochloride in newborn human, 197. *See also* Anterior-posterior tongue areas
Postingestive effects, 11, 19, 101, 164, 328, 329, 357 ff, 367 ff, 410. *See also* Taste aversion, Sham drinking, and Gastric capacity, distension, and loading
Postnatal experience of taste: possible differential effects of, on late maturing and precocial species, 62
Power functions, 289
Preference: measures of, other than intake in ingestion situations, 91; acquired as opposed to genetically determined, 161; for ethanol, 378; effects of illness upon, 404; independence of, from testing experience in weanling rat, 102; for dextrose and sodium saccharin over ethanol, 378, 379; sensory specificity and hunger dependence of, 356; shifts of, 402, 403, 404; transitivity of, 401, 402; tests of, in cortically ablated rats, 200, 201
Preference-aversion function: completion of, with less than isotonic concentrations of saccharin, 365; for sugar, differential survival of, under sham drinking of osmotically matched salt and water, 366, 367; for humans per intake, 369, 370
Preferential discrimination: as opposed to sensory discrimination, 399, 400
Preferential hierarchies, 401
Preference matches: among various sugars, 11
Premastication, 239, 246
Premature human: differential response of, to taste, 128; neonatal pig as a model for, 88
Pre-school children: most preferred snacks of, 253
Protein: in relation to other diet elements, 23; selection of, and age, 23, 338, 339; deficiency of, under sugar feeding regimen in humans, 247; failure of activity to influence selection of, 340; altered selection of, under starvation induced weight loss, 341; defense of minimal percentage intake, 342
Protein starvation: differential effects of, on preferences measured in home cage and brief exposure tester, 410

Quality coding: by cross fiber pattern and labelled line mechanisms, 14. *See also* Specificity
Questionnaire: concerning snack food preferences, 252, 253; concerning appetite, general health, and caries in relation to the frequence of eating specific foods, 254, 258; concerning the food preferences of young adult males, 269 ff
Quinine rejection: in human neonate, 99-101, 174-178; quantification of, per category rating scales in human adult, 285-287; in rat neonate, 101-105; in hamster neonate, 106

Race differences: in preferred level of saltiness and sweetness, 168; as reflected on food preference questionnaire, 270-274
Ratio scales: ratio scaling, 287
Rats: consumption of highest percentage of protein by young, 23; herbivore type teeth and alimentary canal of, 86; wild, 218 ff; intake preference of, for sugar solutions, 387, 398
Receptors, gastric: for sweetness, possible existence of, 358
Repetition of exposure: modification by, of discrimination but not hedonic responses, 404
Respiration: state dependence of, and stimulus effects on in fetus, 111; similarity of pre and post natal stimulus control of, 111; changes of, in response to taste in the newborn human, 127; recording of, during taste testing, 132; disturbance of, in sucrose ingestion by newborn humans, 141, 142
Response profiles: for single fibers based on best of four tastes, 28
Responses of nerves: compared to choice behavior, 198, 199
Retch reflex: maturation of, and oral rejection, 66
Rewards: sweet foods as, 241

Saccharin: taste of, for rats, 42-48; aversion to, in pig, 90; sucking response to, in newborn human, 130; preference-aversion function for, in humans by intake method, 369, 370; butter taste of, and rejection of, in human, 369, 370; preference for, in rat, 404, 405; preference for, over ethanol, 379
Saccharin deprivation effect, 357
Safety of food, 218
Saliva: enzymes in, 246
Salt solutions: aspiration of, by newborn human, 119; graded response to, among human newborns, 129; negative response to in newborn human, 129, 130; failure to detect aversion of newborn human to, 162, 163; responses of newborn human to, 210, 211; maximally preferred concentration of, 167, 168; intensity and hedonic quality of, as judged by the same individuals, 285; maintenance of homeostases for, 414
Satiety: factors influencing, 357-359
Scales of measurement: nominal, ordinal, interval and ratio, 283 ff
Selection, dietary: minor role of sweetness in, 336
Self feeding: transition to, coincides with an increase in the size of the fungiform papillae population on the tongue of the rat, 74
Self-select diet, 23, 409, 411
Seriation of stimulus effectiveness: of sugars, 9, 10, 11, 387 ff; as a function of concentration in fetal, newborn and adult sheep, 55; for acceptance in neonatal rat and hamster, 105, 106; as a function of concentration in human newborns, 208; as part of a research strategy,

387 ff; and preference hierarchies for foods in rat, 401-404
Set point: deviations from, as defined by Nisbett, 301; closeness to, inferred from minimal yearly weight fluctuation in the absence of dietary restrictions, 301; failure of, to effect hedonic judgements of or intake of milk shakes, 302, 303
Seventh cranial nerve, 14, 190, 192, 193; receptor field of, in catfish, 17; integration of, with ninth nerve response function to predict choice behavior, 198, 199
Sex differences: among newborns, 162; in preferred level of sweetness but not saltiness, 168; in adaptation to drops of water, 208; in resistance to the effects of overnutrition in infancy, 314
Sham drinking, 365-367
Snacks: consumption of, for groups differing in caries frequency, 250, 251, 345; relative preferences among, per questionnaire, 252, 253; purchase of, per observational study, 252; strain differences in meal patterns among rats, 348
Social facilitation: of eating in suckling pigs, 95
Social interaction: in the transmission of rat feeding preferences, 230; and aggression at the feeding site in rat, 234
Social setting: role of, in the disinhibition of facial responses to food among mature humans, 184
Socio-economic class: and the form of sugar intake, 241; and obesity, 248
Solid food: rat pups' initial consumption of, 223, 224; transition to a diet of, and changes in the filiform papilla population of the rat tongue, 74-77
Somatotype ponderal index (SPI): in the analysis of questionnaire food preferences, 274
Sour: preference for, 247; effects of, on the response to sweet in human newborns, 162, 163
Spices: in pre-lacteal feeding, 237
Species differences: for sensitivity to sweet (man, rat, hamster and squirrel monkey), 25, 26; in response to substances that taste sweet to humans, 42
Specificity: of taste nerve fiber response, 14, 25 ff; of gymnemic acid effect to sweet taste, 7; of lesion induced deficit to the sweet taste quality, 14
Squirrel monkey: response profiles of single Chorda Tympani fibers in, 27
Stability: of sucking behavior from day to day in newborn humans, 133; of the effect of a glucose load on taste preference in man, 320, 322, 370-372
Starvation: differential effect of, on carbohydrate and protein intake, 341
State: and responsiveness in the fetus, 64; as a determiner of the effectiveness of touch in eliciting sucking, 69; effects of, on sucking to saccharin in the human infant, 130; in relation to hunger, 142; effects of, on some preference measures in newborn humans, 164; physiological, causally unrelated to schedule induced polydipsia, 375; internal, nutritional and chemical, 415
Static phase of lesion induced obesity: comparisons with artificially extended Dynamic phase, 310-312
Stimulus area, 405
Stimulus change: response to, in neonate human, 209
Streptococcus mutans: role of, in caries, 347, 348
Sucaryl: preference-aversion functions for, among human adults by intake method, 369, 370
Suckling-mothering experience: association of, with a specific inhibition of papilla development in rat, 83
Sucking: inhibition of, by bitter, 28; initiation of, by touch and later control of, by taste, 65; failure of, to reflect satisfaction or acceptance, 127; in tests of neonatal taste, 128; taste elicitation of, failure to increase with age in neonate human, 128; elicitation of, by non-taste oral stimulation, 131; computer assisted analysis of, 132; rates of, under no-fluid and 15% sucrose conditions, 136, 137; neglect of amplitude quantification for, by students of newborn human, 146; modification of components of, through changes in nature and concentration of sugar solution delivered, 195-197; disruption of, by quinine hydrochloride in newborn humans, 197
Sucrose: effectiveness in relation to other sugars, 9, 161 ff, 197; preference for, by fasted neonatal pigs in 2-bottle test, 91; effect of, on sucking in human infants, 143; preference for, over formula by newborn humans, 164; as taste stimulus for newborn humans, 174; response to, in human newborns, 196, 197; effectiveness of, in eliciting anterior tongue movements relative to glucose, 197; intolerance of, 243; addition of, to milk shake for highly palatable, highly familiar taste test substance, 299; aversion to taste of, among obese children and obese rats, 324; intake of, and concurrent intake of other nutrients, 330-343; fatal overingestion of, under limited access time feeding schedule, 336-341; other stimulus properties of, 356; preference-aversion function for, from human intake, 369; intake preference thresholds for, in rat, 389, 390; maximal intake of, in rats relative to other sugars, 394, 395; energy value of, in relation to activity and survival in rats, 395
Sucrose free diet: among human neonates, 242; of fructose intolerant individuals, 350
Sugar: addition of, to commercial baby food, 235; addition of, to children's medications and vitamins, 240; in pre-lacteal feeding, 235; ingestion of, and reduced intake of other dietary elements, 258; availability of, and health and growth in rats, 258 ff; intake of, in relation to level of caries, 345; rationing of, and low levels of caries, 345; form of, in determining effect on level of caries, 346; aversion to hypertonic solutions of, in rat, 365; harmful effects of, through distortion of other dietary intake, 395; preference for, in rat, 420 ff; solutions of, stimulus properties of beyond sweetness, 356
"Sugar teat," 125, 240
Superior laryngeal nerve (SLN): recordings from, in newborn lamb, 112, 113
Supplemental feeding of human infants: aims of, 238; trend toward early onset of, 240; in cross cultural perspective, 239 ff
Survival value: of neonatal taste sensitivity, 127; of carbohydrate choice under food deprivation conditions, 341
Swallowing: disruption of, by ablation of vagal lobe in

catfish, 18; in sheep fetus, 57-60; in human fetus, 60, 61; as a possible early manifestation of feeding behavior, 65; in newborn lambs, 111 ff; measurement of, in newborn lambs, 112; pattern of, during sucking in newborn human, 120, 121; insufficient study of, in newborn human, 146
Sweet acceptance: in human neonate, 100, 101, 106; in rat neonate, 101-106; in hamster neonate, 105-106
Sweet foods: preparation of, by spider monkey, 4; use of, as rewards, 241; under representation of, among most highly preferred foods, 270
Sweet substances: inverse relation of appetite for, to appetite for fats, 23; aversion to, among obese humans and animals, 323, 324; other stimulus properties of, 356
Sweetened beverages. *See* Drinks, soft
Sweetness: contrast of, with pleasantness, 293, 294; role of, in determining intake, 356, 357; as a motive for drinking in rat, 356, 357; continuous adjustment in sensitivity to, in the service of regulating nutritive balance, 360; species difference in response to, 25, 26; possible differential exposure to, via fruit drinks, 273; as an unconditioned stimulus for eating and drinking in the hungry organism, 359; subjective reports of aversion to, for both hypo and hyper-tonic solutions, 369, 370
Sweets: eating of, between meals and caries, 250, 251; most preferred snack food by questionnaire, 252, 253

Taboos: feeding, 236
Tactile stimulation: oral, in the initiation of sucking, 68, 69, 131; lingual, in the elicitation of lateral tongue movements, 205
Taste aversion, conditioned: in rat pups, 227; as an example of modulation of palatability by learning, 359; flavor control and non-injection control in demonstration of, 227, 228
Taste buds, scarsity of, in birds relative to other vertebrates, 4; extra lingual, 5, 114, 115; structural maturation of, in sheep and man, 52; papillae sites for, schedule of postnatal appearance in untreated, early weaned and cross-fostered rats, 72-79
Taste preferences: of normal weight and obese children in relation to those of adults, 319
Taste quality: differential response of taste nerve fibers to, 25 ff; array of stimuli for, 26, 27; differential response to, in newborn rat, 105
Taste receptors, 51; lingual, 5, 52, 210, 211; oral but not lingual, 5; extra oral, 17, 117; location of, in relation to acceptance and rejection, 194; functioning of, in sheep during last third of gestation, 55, 56, 57, 61
Taste responsiveness: of normal weight individuals who were overweight, 298
Taste, sense of: comparison of, with other sense modalities, 61, 405, 406
Teeth: susceptibility of, and caries development, 348; cross species comparison of, 86-88
Temporal organization: of sucking, investigational neglect of, 146, 147; of eating in relation to caries, 250, 251, 345, 348, 351; of swallowing in sheep fetus, failure to discern, 58, 59; of behavior in schedule induced polydipsia, 374
Tenth cranial nerve, 14, 22; distribution of receptors innervated by, in catfish, 17
Testing: arrangements for, to separate responses to test substances from responses to diet, 205, 211, 212, 312
Test stimuli: necessity for adequate variety among, for single fiber studies, 26, 27
Thalamus, 17, 19
Thirst: elicitation of, by hypertonic NaCl but not glucose, 22; increments in, and reduced drinking of hypertonic sugar solutions, 365
Thresholds: for detection of sucrose in relation to degree of obesity, 323; for sugar preference per 2-bottle intake test, 389
Tongue: topographical organization of, 6, 8, 16, 194; development of taste papillae on, 70 ff; employment of, by mammals in taste based rejection or acceptance of food, 193; hedonically specific anterior-posterior movements of, in human newborn, 195-198; touch and taste elicited lateral movements of, in human newborns, 205 ff
Transitivity: of preferences, 401, 402
Twins: preference for sweet and salt among, 167, 168
Two bottle preference test: time limited, in squirrel monkey, 9; differential effects of VMH lesions on, 310-312; and factors determining both choice between concentrations and total intake, 335

Up-down method, 405
Urea: response of newborn humans to, 162, 163

Vagus nerve. *See* Tenth cranial nerve
Vegetable and starch: preference for, among Blacks, 272
Ventromedial hypothalamic lesions: and hyperresponsiveness to sweet taste, 297; in rats, 297 ff; differential effects of, on one and two bottle tests of preference for sucrose, 310-312
Viscosity, 212, 213

Water: in pharynx of newborn lamb and apnoea, 116; failure of, to elicit response from tongue in fetal, newborn and adult sheep, 123; responding to, in relation to adaptation, 124; responding to, in newborn infants, 141, 207, 209; provision of, by sweet solutions, 356
Water taste: in sheep, 124; in newborn human, 211, 213
Weaning: and increases in papillae populations on rat tongue, 72-77, failure to find effects of, on final number of papillae, 75; hazards for wild mammals during, 217, 218; accomplishment of, with the aid of bitter substances in humans, 247
Weanling pigs: food intake responses of, to exogenous insulin, 94
Weight gain: as a measure of 6 hour food intake in piglets nursing sows, 94
Weight loss: failure of, to disrupt preference for saccharin, 379; failure of, to effect sucrose preference in humans, 320, 321

Zucker rats: and genetically determined obesity, 312 ff

☆U.S. GOVERNMENT PRINTING OFFICE: 1977 O—213-143

DATE DUE

MAY 11 1978

APR 30 1985

GAYLORD PRINTED IN U.S.A.

Library
National Institutes of Health
Bethesda, Maryland 20014

New Book 4-10-78

LIBRARY
NIH
Amazing Research.
Amazing Help.

http://nihlibrary.nih.gov

10 Center Drive
Bethesda, MD 20892-1150
301-496-1080

NIH LIBRARY
4 0124 9706

NIH LIBRARY
3 1496 00174 8139
JUN 1 1985
JUL 20 198
FEB 5 198
SEP - 1
AUG - 5

DHEW Publication No. (NIH) 77-1068

1977